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ABSTRACTS OF TUBERCULOSIS

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Tuberculosis in Early Infancy.—Tuberculosis in infancy, being always acquired, is usually acquired through infected sputum, whether this be in a dry or moist state. The amount, even of powdered bacilli, necessary to infect, is very small. Milk, the far less important source of bacilli, is practically never infected in human beings, though not infrequently so in cows. Tubercle bacilli can infect the body by way of the respiratory tract, digestive tract, and skin. Massive infection giving rise to generalized tuberculosis is rare. Next to the alveoli and terminal bronchioles, the commonest site of the initial tubercle is in the upper respiratory passages, that is, the nose and pharynx. Local lesions secondary to dentition and ulcerative stomatitis favor the entry of the bacilli. The ocular mucous membranes must be considered. The frequency of involvement of the digestive tract and of the mesenteric nodes is well known. According to Cohnheim's law, there is always a tuberculous lesion at the point where the tubercle bacillus enters the body. According to Parrot's law, tracheobronchial adenitis is always secondary to pulmonary involvement. Later this was applied to cervical and to mesenteric nodes, to which latter application there are many exceptions. Calmette and Bartel urge a rather different manner of dissemination of the bacilli, namely, by leucocytes in the blood and lymph streams, thus bringing immune reactions into play. The following summarizes the author's classification: *Symptoms, lesions and course of infantile tuberculosis:* The time between ingestion of tubercle bacilli and positiveness of the skin test varies between 6 days and 4 months, being in most cases from 20 to 30 days. During this period the patient is symptom-free. *Early pulmonary stages:* Acute diffuse (nodular or confluent) tuberculous alveolitis. Primitive pulmonary tubercle. (The symptoms of this stage and of the stage of involvement of the tracheobronchial nodes vary greatly.) *Tuberculosis of the bronchial nodes:* First stage: gray infiltration, with many tuberculous follicles but slight degeneration (readily cured). Second stage: caseation (recovery doubtful). Third stage: (rarely encountered) abscess formation (recovery impossible). *Symptoms and varieties of tuberculous tracheobronchial lymphadenitis:* 1. "Silent or florid," revealed only by skin-test, progressing to clinical cure. 2. "Manifest," with subfebrile state, mild bronchitis, loss of weight or failure to gain, varying degree of anemia, moderate diarrhea. 3. Cases with general symptoms, without local manifestations.—*La tuberculose dans la premiere enfance, A.-B. Marfan, Nourrisson, May, 1924, xii, 153.*—(*P. J. W.*)

Tuberculosis in Infancy.—Of 2,300 Pirquet and intracutaneous tests done on infants at the Toronto Hospital for Sick Children, 39 have been positive, a percentage of 1.7. Twenty-nine positive tests have been obtained in infants attending the chest clinic. The period covered is three years. Of all these cases, 45 have been followed for longer than six months. Of these 45, 20 have died, a total mortality of 44 per cent. The mortality for the first year is 70 per cent (14 cases); that for the second year is 24 per cent (6 cases). Prior to the introduction of the skin test for tuberculosis, children were regarded as tuberculous only when they presented clinical symptoms. This explains the very high mortality figures previously obtained. Under one year of age it is still a dangerous infection, with the chances against recovery. Once this age is past, it is not the dangerous infection it was previously considered to be. In 77 per cent of the author's positive cases, there

was definite history of exposure to tuberculosis. The necessity of early separation of infants from tuberculous individuals is clear. —*Tuberculosis in Infancy. Its Prognosis and Prevention, G. A. Davis, Arch. Pediat., March, 1924, xli, 200.*—(*P. J. W.*)

X-ray Studies of Tuberculosis in Infancy.— In the infant tuberculous changes are much more massive than in the adult. Reactions are more intense, and a definite predilection of the lymphatic structures to disease confuses exact interpretation of actual morbid changes. Acute serous changes predominate. Chronicity is the exception. Changes fall into two headings: (1) those involving the lymph nodes and (2) those attacking the parenchyma. The lymph-node changes may be paratracheal, perihilar, or peribronchial. The paratracheal nodes are involved earliest in life, the perihilar somewhat later, and the peribronchial latest. In the early stages, the X-ray shows an evenly outlined shadow in the upper mediastinum, rounded or elongated, depending upon the degree of congestive reaction and the number of nodes involved. Smooth contour and unilateral shadows of even density characterize this stage. If the process is perihilar or peribronchial the changes apply to these respective regions. Differentiation from nontuberculous processes at this stage is difficult, and only subsequent events can clear up the diagnosis. The second stage of changes is that due to necrosis and fibrosis. Outlines are irregular and blotchy, and so called mottling is present. Occasionally one may notice a zone of parenchymal congestion, and when these various changes are present simultaneously the diagnosis of tuberculosis is probable. The ultimate outcome of these changes has been twofold: in one type, a retraction of shadow takes place, with an increase in density, and an accentuation in the mottling; in the other type, a definite progression to parenchymal involvement, with a picture which falls under the heading of mixed parenchymal tuberculosis. Parenchymal tuberculosis may be pleural or pulmonary. The pleural changes are of two types: *1:* An interlobar effusion usually between the upper and middle lobes. It generally subsides leaving a definite transverse, linear scar. *2:* In this type there are either serous or fibrous deposits. The serous deposition is usually in small quantities. The fibrous deposition takes place in the form of fine filament-like bands or, in rare instances, a diffuse, thick, plastic mass, not unlike that seen in the adult pleuritis. A primary uncomplicated tuberculous serous or fibrinous pleurisy has not occurred. The pulmonary changes are of three types: (1) bronchitic (2) bronchopneumonic or lobular and (3) lobar. In the bronchitic the film shows the markings accentuated and somewhat congested in appearance. In the bronchopneumonic type the whole lung may be so riddled that few areas of normal lung are distinguishable. Every conceivable pulmonary change may be shown—small cavities, lymph nodes, large areas of amalgamated pathological processes, and occasionally small areas of spontaneous pneumothorax. In the lobar variety the picture may be indistinguishable from an effusion. Annular shadows may be present. *Conclusions:* Tuberculosis in infancy is of two types, acute and chronic: the first is the more common of the two and may be subdivided into (1) tuberculosis of the lymph nodes and (2) tuberculosis of the lungs. In both cases, by means of roentgen-ray follow-up, the process can be shown to have a definite and fairly orderly sequence, a period of congestion, a period of retraction or spreading, complete or incomplete, and a period of softening. While it is true that in the early or congestive stage the process may not be distinguishable from any of the acute infections, in the second stage of retraction or expansion the condition assumes characteristic appearances, and in the third stage of anatomic changes there should be no difficulty in diagnosis. For exact visualization of these changes, repeated and roentgenographic investigation is absolutely essential.—*A Roentgenological Study of Tuberculosis of Lungs and Intrathoracic Glands in Infancy and Early Childhood, I. E. Liss, Am. J. M. Sc., September, 1923, clxvi, 396.*—(*B. T. McM.*)

Tuberculous Pulmonary Cavities in Infancy.—Following a review of the literature on pulmonary cavitation in infants up to two years of age, the authors present a case of tuberculous cavity in an infant two years old. The patient was a colored boy. The principal symptoms were fever, cough, and dyspnea. The total duration of the sickness was 3 months. He had had pertussis six months previously. Physical examination, X-ray and autopsy all revealed cavitation in the right upper lobe. Additional autopsy findings were miliary tuberculosis of the spleen and liver, tuberculous adenitis, slight bronchopneumonia, fibrinous and fibrous pleuritis.—*Tuberculous Pulmonary Cavities in Infancy, D. M. Siperstein and Harry Bauguess, Am. J. Dis. Child., June, 1924, xxvii, 586.*—(*P. J. W.*)

Transmission of Tuberculous and Syphilitic Antibodies from Mother to

Child.—In 6 women, tuberculosis suspects, the serum reaction (complement-fixation test) was negative (only one was clinically tuberculous). Blood obtained from 3 of their newborn babies was negative. In 8 definitely tuberculous women the serum reaction was positive. The blood of 7 of their children, or of their umbilical cords, gave positive tests (these babies' skin reactions were negative). Five infants gave positive serum reactions with negative skin reactions. These retained their antibodies for only a relatively short time, between one and three months. In known tuberculous women there is a certain transmission of antibodies to their infants; and infants having inherited tuberculous antibodies from their mothers, and having a negative Bordet-Wassermann reaction and a negative skin reaction, retain their antibodies for a short time only.—*La transmission des anticorps tuberculeux et des anticorps syphilitiques de la mere à son enfant, M. L. Ribadeau-Dumas, Nourrisson, May, 1924, xii, 175.*—(*P. J. W.*)

Autopsy Statistics of Tuberculosis of Infants and Children in Japan.—The pathological examinations made at the Tokio Imperial University during the 24 years prior to 1910 are reviewed. There were 1,357 autopsies under 15 years of age, of which 36.85 per cent were proved to by tuberculous. Of these, pulmonary tuberculosis, as well as respiratory lymphatic involvement, numbered 95.6 per cent, the involvement of other organs being small. There were but few cases of intestinal tuberculosis, only 1 per cent of all the tuberculous cases.—*Pathological Statistics on the Tuberculous Foci in Postmortem Examinations of Infantile Cases, T. Sato, Kekkaku July, 1923, i, no. 4, abstracts from Japan Medical World, February, 1924, iv, 45.*—(*M. M.*)

Death from Miliary Tuberculosis Forty-one Days after Birth.—In this case death occurred on the forty-first day after delivery. The difficulty of deciding by which route the baby was infected is emphasized. The baby was born by Caesarean section and was normal in every way. The mother had a fever of 101.6° on the afternoon of delivery. She died of miliary tuberculosis forty-four days later. The baby developed fever (105°) at the age of one month, dying in 10 days. Autopsy revealed generalized tuberculosis, especially in the lungs, spleen, and liver. The mesenteric lymph nodes showed almost complete caseation and necrosis. The possible channels of infection were germinal, intrauterine, intrapartum, and neonatal. It is of interest that three examinations of the mother's sputum were negative.—*A Case of Tuberculosis in Infancy, N. B. Capon, Brit. J. Child. Dis., October–December, 1923, xx, 209.* (*P. J. W.*)

Tuberculous Pyopneumothorax in Infant.—The father had open tuberculosis and lived with the family in cramped quarters. The baby was admitted at the age of 3 months, suffering from cough and loss of weight. Intracutaneous tuberculin test, negative at first, later became positive. At the age of 5 months, he had an attack of acute dyspnea with violent coughing and cyanosis, and all the physical signs of left pneumothorax, corroborated by X-ray. He died eight days later. Necropsy revealed the left lung collapsed, with two perforations in the visceral pleura near the midlateral aspect, these communicating with a pulmonary cavity. The left lung was so compressed and diseased that the division of the upper and lower lobes was lost and the lung fused with the mediastinal tissues. The bronchial nodes were completely caseous. There was advanced caseation in the central part of the lung. It is of interest that during the last illness of the patient there was no fever.—*Tuberculous Pyopneumothorax in an Infant Aged Five Months, S. G. Ross and C. T. Crowdy, Am. J. Dis. Child., May, 1924, xxvii, 456.*—(*P. J. W.*)

Severe Tuberculin Reactions in Children.—One hundred and seventy boys in an industrial school near Edinburgh were examined because of occasional outbreaks of sudden, severe illness. The Pirquet test was positive in 101 boys, or in about 60 per cent. There were 5 cases giving extremely severe reactions, the long diameter measuring between 35 and 40 mm. Three showed a large central vesicle. In another industrial school in the same area, only 14 per cent of the tests were positive. The boys in the former school showed a high incidence of chronic conjunctivitis, nasal catarrh, and phlyctenular disease. The most severe reactions are commonly associated with these conditions. Such cases of "scrofula" are apparently associated with status lymphaticus, which results in intensified reaction to the tubercle bacillus and its poisons.—*Severe Tuberculin Reactions, Scrofula, and Status Lymphaticus, C. McNeil, Brit. J. Child. Dis., October–December, 1923, xx, 197.*—(*P. J. W.*)

What the Tuberculin Skin Reaction Has Taught.—The tuberculin skin test has definitely established the fact that the incidence of tuberculous infection increases

with age (whether such infection be latent or active). Such infection is almost never congenital. It is contracted usually during the first few years of life. The first infection immunizes the majority of individuals. What is considered an hereditary predisposition to tuberculosis is the result of a familial contact. Immunity to tuberculosis is most commonly acquired between the ages of one and fifteen. This immunity is elaborated principally by foci in the lymph nodes. Children failing to be thus immunized, succumb to the more or less continual progress of the first infection. Immunization by first infection is not absolute and varies in degree. The question of *superinfection* (superimposed, later infection) or of activation of an old infection by loss of immunity, is not yet solved. It is now clear that in a few infants under one year of age, tuberculosis may pursue a relatively benign course. Hereditary transmission of immunity is apparently a factor in such cases. Removal from contagion is particularly important during the first two years of life, because immunity is acquired with such difficulty at that age. Finally, the author believes that it is rarely possible to establish the fact of a new infection in an adult already infected since early childhood. Autoinfection from the old focus as a result of loss of immunity is more likely.—*Les enseignements de la cuti-réaction—l'immunité anti-tuberculeuse, A.-B. Marfan, Nourrisson, March, 1924, xii, 73.—(P. J. W.)*

Value of Tuberculin Skin Test.—A positive skin reaction indicates that the patient is infected with living tubercle bacilli, but does not indicate that he is suffering from an active tuberculous process. Reactions are classified as (1) severe, usually early, (2) faint, (3) delayed. A skin reaction may be negative because the individual does not harbor the tubercle bacillus, or because he has lost the power to resist the tubercle bacillus (anergy). The author distinguishes (1) anergy of the period of incubation of tuberculosis, (2) anergy due to extension of the tuberculous lesions, accompanied by cachexia, (3) anergy associated with menstruation, pregnancy, confinement, lactation, (4) anergy associated with pneumonia, grippe, whooping-cough, cirrhosis, and (5) anergy during intensive treatment with tuberculin. The skin reaction remains the ideal test for activity of tuberculosis. —*La cutiréaction à la tuberculine—sa valeur pour le diagnostic de la tuberculose des enfants du premier age, A.-B. Marfan, Nourrisson, January, 1924, xii, 9.—(P. J. W.)*

Serum Diagnosis of Tuberculosis in Children.—In a number of diseases the albumin-globulin balance is altered in such a way that the abnormality can be detected readily by treatment of the serum with ammonium sulphate. The method is particularly applicable to the small amounts of blood obtainable from children. One-tenth cc. of serum is treated with 5 drops of 19 per cent ammonium-sulphate solution. If a definite flocculation develops in 5 minutes the reaction is considered positive. Serums from cases of active tuberculosis and syphilis gave positive reactions. In fevers of other etiology and in suppurative processes the reaction was always negative.—*Zur Serodiagnose der Tuberkulose im Kindesalter, F. Mündel, München. med. Wchnschr., February 1, 1924, 132.—(E. R. L.)*

Complement Fixation in Tuberculosis of Children.—The patients on whom serological studies have been made are grouped as follows: (1) anergic, cachectic, very advanced bilateral pulmonary tuberculosis; (2) pulmonary tuberculosis with general condition fair; (3) unilateral pulmonary tuberculosis treated by artificial pneumothorax; (4) tracheobronchial adenopathies; (5) pleural and peritoneal tuberculosis (with tracheobronchial adenopathies). From the statistical tables presented, the complement-fixation reaction is shown to be positive in patients found to be definitely tuberculous. Patients with negative reactions have given positive reactions when tested again later. It is often negative in the very mild cases, which, however, give a strongly positive Pirquet test. There was practically no agreement between the intensity of the Pirquet test and the degree of fixation of complement. The complement-fixation test is merely one additional finding, to be considered in connection with clinical, radiological, and laboratory observations. To date, the reaction possesses no definite significance.—*Contribution à l'étude des anticorps dans le sérum des enfants tuberculeux, Armand-Delille, Isaac-Georges and Ducrohet, Arch. de med. d. enf., February, 1924, xxvii, 94.—(P. J. W.)*

Detection of Tuberculosis among School Children.—By systematic examination the tuberculous suspects among children are sorted out and are then further examined. Definite local and general symptoms lead to the suspicion of tuberculosis. Phlychtenulae, tuberculids, joint and bone caries, and scrofulosis are the more frequent conditions. In addition, are to be con-

sidered fibrous lesions of the cornea and affections of skin, bones, etc. Nonspecific symptoms are catarrh of the nasopharynx, indurated lymph nodes extending to the clavicle, in the axilla, and rarely on the sides of the chest, and pallor of skin and mucous membranes. About 5 per cent of the children were thus sorted out.—*Wie erfasst man die Tuberkulose in der Schule? Planner, Beitr. z. Klin. d. Tuberk., May, 1923, lv, 149.*—(*H. J. C.*)

Tuberculosis of Childhood.—Tuberculosis of children between the ages of 5 and 12 is essentially a disease of the lymphoid tissue. In order to understand the course of tuberculosis at this period it is necessary to know the thoracic lymphatic system. In the lungs there is a deep and a superficial set of lymphatics. The two systems are connected and their afferent trunks converge in the lymph nodes of the hilum. The mucous membrane of the lungs is in direct contact with the outside world through the inspired air. Any bacteria that may reach the air cells in this way may be conveyed into the lymph spaces. The lymph stream then carries them to the tracheobronchial nodes. The lymph drainage of the intestinal tract and the cervical region, eventually through the subclavian veins, the pulmonary artery and lung capillaries, get to the tracheobronchial nodes. These nodes are, therefore, receiving stations for tubercle bacilli no matter how they enter the body. In children the place to look for early tuberculous disease is in the lymphoid tissue about the trachea and larger bronchi. When this group of nodes alone is involved the condition is best defined by the term bronchial adenitis. The next step in the progress of juvenile tuberculosis, after involvement of the tracheobronchial nodes, is to affect more of the pulmonary bronchial group at the root of the lung, and peribronchial tuberculosis develops. At this stage, in addition to the fatigue, listlessness, undernutrition and irritability, present in the bronchial adenitic stage, there are more constitutional and local symptoms present, such as cough, low fever and local sweating. The X-ray film often reveals the first visible focus of pulmonary tuberculosis in the second interspace when no signs have been found on physical examination. The transition from hilum tuberculosis to pulmonary or adult type takes place in three different ways. The most common is by direct extension outward into the pulmonary tissue. The second is by rupture into a vein causing general dissemination of tubercle involving both lungs. The third is by ulceration of a caseous focus into a bronchus; the whole section of the lung tributary to this bronchus may be thus involved, and a condition of tuberculous bronchopneumonia develop, as a result of massive infection. Hilum tuberculosis is a comparatively benign infection. Pulmonary tuberculosis in children is a malignant disease.—*The Evolution of Juvenile Tuberculosis, H. D. Chadwick, Boston M. & S. J., August 7, 1924, cxci, 252.*—(*B. T. McM.*)

Tuberculosis in Children.—The comparison, at intervals, of weights and measures is the best indication of a child's growth and, therefore, of its health. Eight per cent of underweight children have evidence of tuberculosis. Pasteurization of milk cuts down the incidence of bovine infection. Fatigue is a prominent symptom of early tuberculosis in childhood. Cough is not so significant a symptom as fatigue. A temperature of 99.4°F. is normal for a child. The best treatment is routine sanatorium treatment.—*Tuberculosis in Children, H. D. Chadwick, Boston M. & S. J., July 10, 1923, cxci, 58.*—(*B. T. McM.*)

Primary Lung Focus in Tuberculosis of Children.—As a result of the examination of 347 children at postmortem, of which 229 revealed primary foci and 205 primary complexes, the following conclusions are drawn: The hematogenous route for the origin of the primary focus in the lung is denied since a tuberculous lesion in other parts of the body was never discovered and not considered tenable, it being questioned that a hematogenous seeding would lead to the development of a single or a few individual foci. Also, the retrograde lymphogenous origin of the primary focus from the regional bronchial lymph nodes may be excluded with certainty. In the majority of cases the first expression of childhood tuberculosis is the primary focus of the lung originating from inhalation. From this source in relatively short time there results infection of the regional lymph nodes producing the primary complex. In addition, this material tends to show the great tendency of the primary infection to heal.—*Der primäre Lungenherd bei der Tuberkulose der Kinder, M. Lange, Ztschr. f. Tuberk., May, 1923, xxxviii, 167.*—(*H. J. C.*)

Healed Miliary Tuberculosis in Child.—Evidence of repeated infection from the original tuberculous focus is found in the presence of crops of tubercles of different sizes. The focus of dissemination may be so small that the number of tubercle bacilli discharged may not produce a severe infection, and healing of the focus may take place.

Under such conditions, recovery may occur. The patient presented is a boy of eleven, who had had attacks of dyspnea since the age of one year. Tonsils and adenoids were removed at the age of five At eight years he had pertussis, at ten influenza followed by pneumonia. He was brought to the hospital because of asthmatic attacks, and remained there five days. The temperature was normal. Intracutaneous tuberculin test (0.1 mgm.) was negative. X-ray showed small calcified areas scattered throughout both lungs. There was no special accumulation of these in the vicinity of the roots. After four years there was no change in the X-ray. The asthmatic attacks were decidedly infrequent.—*Report of a Case of Healed Miliary Tuberculosis, R. W. Baer, Am. J. Dis. Child., February, 1924, xxvii, 110.*—(*P. J. W.*)

Clinical Observations on 58 Cases of Tuberculous Meningitis.—Statistics of the incidence of all the various signs and symptoms of this condition in 58 cases are given in detail. It is of interest that Kernig's sign was positive in only 27 per cent, and that the tuberculin reaction was positive in 86 per cent and negative in 14 per cent. The mortality was the usual 100 per cent.—*Considerazione cliniche su 58 casi di meningite tubercolare, S. Fabris, Pediatria, February 15, 1924, xxxii, 199.*—(*P.J.W.*)

Mortality of Pulmonary Tuberculosis in Children.—Of 100 cases of open pulmonary tuberculosis among children 72 had died within 10 years, and 9 had an unfavorable prognosis. This agrees with the figures of Simon (*Ztschr. f. Tuberk., 1917, xxviii*), and Hahn and Geissler (*Dissertation, 1918*) on the generally unfavorable course of pulmonary tuberculosis in children.—*Das Schicksal offen tuberkulöser Kinder, Geissler, Beitr. z. Klin. d. Tuberk., May, 1923, lv, 155.*—(*H. J. C.*)

Treatment of Bone and Joint Tuberculosis in Childhood.—The conservative method of treatment is preferred. This is departed from only in special cases, when financial conditions demand a shortening of the treatment or when an increasing destruction indicates operative intervention. The favorable outcome of childhood tuberculosis, as well as the multiple lesions, make operative treatment inadvisable. The fresh-air treatment is carried out as far as possible even at night, and rest among restless children is enforced by plaster casts and straps. In tuberculosis of the skin, lymph nodes and peritoneum the roentgen ray is used with benefit. In bone and joint tuberculosis judgment is reserved as to its value. Stasis and iodides yielded no results; on the other hand the limited movements of Bier are recommended. Specific treatment is discarded.—*Zur Behandlung der kindlichen Knochen und Gelenktuberkulose, S. Simon, Arch. f. klin. Chir., 1923, cxxiii, 807.*—(*H. J. C.*)

Prophylaxis of Tuberculosis in Children.—Tuberculosis in children has markedly increased in incidence and severity. Tuberculin is of greatest significance in diagnosis; in the early years of life a positive reaction speaks for active tuberculosis. The Pirquet test holds first place. The duty of the preventorium and dispensary is to properly treat tuberculous children. Carbohydrates should be restricted for such children, while proteins and fats should be increased, especially when carbohydrates had been greatly taken previously. Sufficient clothing is important, and overheating is to be avoided in winter. *Tebelon* is not indicated in advanced cases, the most favorable period for its use being 2 months after beginning treatment. Every tuberculous infection orginates from a phthisical individual. For determining tuberculosis in children the finding of bacilli does not play the same rôle as in adults, but tuberculin testing cannot be dispensed with. In the nursing child every infection is practically fatal. During early childhood every positive-Pirquet child is ill and requires treatment, while during the school ages treatment is necessary only when the suspect is definitely tuberculous.—*Die Kinderfürsorge in dem Tuberkulosefürsorgestellen, W. Stoeltzner, Ztschr. f. Tuberk., September, 1922, xxxvi, 569.*—(*H. J. C.*)

Prophylaxis of tuberculosis in Childhood.—Children with subnormal resistance who grow up and become tuberculous in later years furnish one of the chief sources of adult tuberculosis. Inasmuch as by appropriate treatment a subnormal immunity may be more or less corrected it is of the greatest importance to investigate the immunity of children and to employ suitable measures for the treatment of all who prove to be suspects. It may be accepted as a preliminary that patients of a tuberculous type with a low hemoglobin content almost certainly have a low threshold of resistance to tuberculosis. By investigation of the tuberculous immune bodies according to Spengler's methods a fairly certain diagnosis might be made for all doubtful cases. Measures to prevent tuberculosis by diagnosis and treatment of subnormal immunity during childhood would of necessity be on a scale so immense that to be thorough they

must be undertaken by the State. They would eventually be a source of wealth through their effectiveness in eliminating tuberculosis and improving the health of communities.—*The Problem of the Tuberculous and Pretuberculous Child, R. Simpson, Practitioner, 1924, cxii, 193.*—(*E. C.*)

Blood Counts. Erythrocyte-Precipitation test and Prognosis of Tuberculosis in Children.—The material for this study consisted of 150 children with various types of tuberculosis, and from this group 10 case reports are cited as typical of the findings. It would seem that an increased rapidity in the sedimentation of erythrocytes, as shown by the citrate test of Linzenmeier, and especially in conjunction with a lymphopenia and gradual disappearance of the eosinophiles coincidently with an increase of the neutrophiles, is of bad prognostic import regardless of clinical findings. Conversely, a normal precipitation reaction and normal blood picture indicate a good prognosis.—*Die Bedeutung der Beziehungen Zwischen Blutbild und Erythrozytensenkungsgeschwindigkeit für die Prognosestellung der Tuberkulose bei Kindern, K. Gulden and E. Lüders, Arch. f. Kinderhk., 1924, lxxiv, 145.*—(*T. C. H.*)

Prognosis and Treatment of Acute Serofibrinous Pleurisy in Children.—Here we have to deal most often with a pleuropulmonary rather than purely pleural disease. Although certain acute infectious diseases may give rise to pleurisy with effusion the most frequent cause is, as with adults, tuberculosis. In childhood, however, there are graver possibilities which must not be ignored. It may be but a manifestation of a generalized tuberculosis and commonly is associated with tracheobronchial adenopathy. *Immediate prognosis:* Generally in infants under two years we are dealing with a generalized tuberculosis, and the prognosis is grave. In somewhat older children, for example four or five years, we may have a generalized serous membrane tuberculosis, a pleuroperitonitis, and a meningitis may terminate things. However, certain cases are cured. Finally, there are cases representing an isolated manifestation confined to the pleura which get entirely well. This is more apt to occur in older children, over thirteen or fourteen years, when the type approaches that of the adult, but may occur also in those from five to ten years of age. In the truly primary pleural tuberculosis a single aspiration usually suffices. In all cases attention should be paid to persistence of the fever, and possible pulmonary manifestations. The volume of the effusion, and still more, the rapidity of its accumulation, are important, because sudden death from asphyxia or cardiac failure may occur within a few hours, unless relief is had by thoracentesis. Such fatalities are more common with right-sided than left-sided effusions, in the proportion of 2 to 1. The reason lies in the compression of the base of the heart, including the great veins and right auricle, leading to death from syncope. Large right-sided effusions should, therefore, be aspirated without delay. On the other hand, with effusions in the left pleura, when the heart is first displaced, the apex moves downward. This does not embarrass the circulation until a point is reached where torsion occurs. It is also easier to determine the size of left-sided effusions, because of the displacement of the heart apex and obliteration of Traube's semilunar space, while on the right considerable effusions may exist in the mediastino-diaphragmatic region without detection. Regarding the frequency of sudden death in children with effusions, Israel finds only 2 out of 206, one due to paralysis of the heart by compression, the other to pulmonary embolism following thoracentesis and lavage. The heart is the important factor; it must be carefully watched, and if it seems weak aspiration must be done. *Ultimate prognosis:* The future of pleuritic children is extremely variable, some developing an acute tuberculosis; others, none at all. Others gradually manifest secondary parenchymatous changes. Many cases, especially in the latter part of childhood, recover completely, and it is certain that many subjects with a pleuritic history never become manifestly tuberculous. To set an arbitrary standard, one should not speak of a complete cure unless at least three years have passed without secondary manifestations. If a pleurisy is entirely cured, there should be complete apyrexia, no acceleration of the pulse rate, and the child should gain weight. Otherwise one should look for a development of disease of the lungs. A cortico-pleuritis may exist for some time, yet finally disappear, and probably confers some degree of immunity. *Treatment:* First of all comes rest. Dry cupping and mustard plasters may be applied. Calcium chloride in 10 or 12 grain doses in a suitable vehicle (syrup of orange or lemon) is thought to hasten absorption. Autoserotherapy is mentioned to be condemned because of the frequent presence of tubercle bacilli in the fluid. Of great importance, when there are functional cardiac disturbances, cough, dyspnea, and a tendency to asphyxia, is thoracentesis by means of the apparatus of Potain or Dugnet. One evacuates the fluid slowly

and not completely, or else replaces it by air or nitrogen, according to the method of Achard and Grenet, or Vaques and Touraine. Forced reëxpansion of the lung may lead to acute congestion and albuminous expectoration; hence the value of injecting gas as the fluid is withdrawn. *Sequellae:* Postpleuritic scoliosis or thoracic deformity should be treated by respiratory exercises and suitable muscular or gymnastic exercise or reëducation.—*Pronostic et traitment de la pleurésie aigüe séro-fibrineuse de l'enfant, M. P. Lereboullet, Prog. Méd., March 8, 1924, no. 10, 153.*—(*A. P.*)

Respiratory Function in Pulmonary Tuberculosis.—The authors have made a study of the vital capacities and the maximum volume of air respired per second in various clinical forms of tuberculosis in process of evolution. The observations were made upon 55 male patients, aged 20 to 55 years. An additional 4 cases of artificial pneumothorax were studied. The authors go into considerable detail and conclude as follows: 1. Study of maximum volume of air respired, per unit of time and of vital capacity, furnishes information of the greatest clinical interest in tuberculosis, provided the investigations are carried out in series in the same patient. 2. During the evolution of fibrocaseous pulmonary tuberculosis (the common form with its various clinical manifestations), the maximum respiratory volume per unit of time and the vital capacity undergo progressive parallel diminution during an intercurrent exacerbation. This fall is in proportion to the acuteness of the trouble; they both rise again slowly and progressively during resolution of this exacerbation and during the period of remission which follows. A return to the figures noted before the extension takes place but slowly, and never completely as regards vital capacity. 3. In acute pulmonary tuberculoses (caseous pneumonia, caseating pleural pneumonia and tuberculous septicemia) maximum volume per unit of time and vital capacity fall rapidly and markedly in proportion to the acuteness of the process. 4. In fibrous pulmonary tuberculosis, diffuse and with emphysema, the maximum volume per unit of time undergoes a reduction which is marked, while the vital capacity shows only a slight reduction; at the same time both respiratory volume and vital capacity fall simultaneously with outbreaks of intercurrent bronchoalveolitis and pulmonary congestion. 5. During the establishment of artificial pneumothorax, maximum volume per unit of time, after a period of suspense of variable duration, recovers progressively in proportion as the lung is collapsed. Inversely, the vital capacity during this period diminishes slowly. Pneumothorax once accomplished, the curve of maximum respiration volume per unit of time rises, to attain its precollapse figure after two or three months and even to exceed it during the months which follow. The curve of vital capacity continues to fall slowly for several months and does not reach the neighborhood of its point of departure until after a year or so. 6. Variations in maximum respiratory volume per unit of time, and those of vital capacity, observed in the course of evolution of pulmonary tuberculosis appear to be determined mainly by the acuteness of the tuberculous process. Under certain circumstances, notably during the recovery of respiratory capacity in the course of therapeutic pneumothorax, the unit-time respiratory volume and particularly the vital capacity appeared influenced, the first by the creation of a vicarious emphysema and the second by the functional adaptation of the thoracic box. 7. The manometric mask of Pech and the spirometers, through the information which they give concerning the evolution of pulmonary tuberculosis, and by virtue of the ease with which they may be handled, warrant their use in the future in the practical treatment of tuberculosis.—*La fonction respiratoire au cours de l'évolution de la tuberculose pulmonaire (formes cliniques, pneumothorax artificiel); débit respiratoire maximum et capacité vitale, M. Piéry and H. Boisseau, Ann. d. Méd., Par., October, 1923, xiv, 284.*—(*W. B. S.*)

Systemic Blood Pressure in Hemoptysis.—Although an unduly high blood pressure has commonly been regarded as a predisposing cause of pulmonary hemorrhage, there is little evidence to support such a view as far as phthisis is concerned. Even factors causing temporary rise, such as overexertion, excitement, etc., seem to have little if any etiological significance. Bang found that in practically 70 per cent of 2,000 patients the initial hemoptysis came on while they were reclining, and in only 6 per cent while they were walking or working. Several observers, notably Grant and Pottenger, have noted a characteristic decline in blood pressure with advancing disease, especially in the diastolic pressure. Waller has recorded careful observations on 50 hemoptysic subjects. In 76 per cent there was a definite decrease in systolic tension which bore little relation to the amount of blood lost. In every case the diastolic index was lower than normal; that is, less than two-thirds the systolic pressure, and in 58 per cent reduced to one-half or less of the latter. This suggested the taking of similar

observations on 25 patients undergoing tuberculin treatment, and one could not but be impressed by the general trend of the readings and indices downwards after inoculation of tuberculin, the diastolic tension being reduced after injection in 88 per cent. Observations were then made on 25 phthisical patients subject to intermittent pyrexia, with manifest toxemia. The extremely low figures registered were noteworthy, the diastolic pressure again being reduced more than the systolic. To summarize, it may be said that in the toxemia of phthisical patients, however brought about, there is an associated lowering of arterial tension and tonus, manifested by a change from Faught's normal 3:2:1 (systolic, diastolic, and pulse pressure) ratio to one of 3:1.5: 1.5 or 3:1: 2. As a corollary, hemoptysis may be regarded as evidence of heightened activity of the lesion, at least temporary.—*The Relationship and Significance of Systemic Blood Pressure in Hemoptysis Due to Pulmonary Tuberculosis, P. S. Walker, Tubercle, February, 1924, v, 201.*—(*A. P.*)

Blood-Cell Volume in Tuberculosis.—The chronic ulcerative type of pulmonary tuberculosis is quite commonly accompanied by, or rather, responsible for, a disintegration of certain blood elements. When the cell count and hemoglobin failed to help in judging the condition of a patient, it was found that volumetric data were always significant and equally consistent for the same persons. The significance of the factor of relative corpuscular volume has not been emphasized heretofore. The volumetric determination of the blood's corpuscular content by centrifugating oxalated blood is an old method and quite reliable. Introduction by mouth of large volumes of water into normal persons alters the blood concentration little or not at all. It is the nature of the body to maintain its actively circulating fluid, the blood, in constant proportion. The method is as follows: 10 cc. of blood is withdrawn from a vein and added immediately to 2 cc. of 1.6 per cent sodium-oxalate solution in a graduated 15 cc. centrifuge tube. The tube is inverted three times to thoroughly mix contents, stoppered with cotton or cloth and centrifugated at high velocity for half an hour or longer. The graduated tube of the Sahli hemoglobinometer can be used for the work, in which case 2 cc. of blood is added to exactly 0.4 of oxalate solution. The corpuscular deposit is subtracted from the total blood inserted in the tube, and the percentage by volume calculated. With the method it was found that the relative blood-cell volume was fairly constant for the same person over a period of time. Healthy or normal men gave relative corpuscular volumes of from 44.5 to 48 per cent. Forty tuberculous men were then examined and were found to fall into three groups. *Class A* gave a relative corpuscular volume of above 48 per cent, ranging from 49 to 52.4 per cent. The figures for *class B* coincided with those for the healthy men. *Class C* was the group below 44.5 per cent, with some as low as 40 per cent. A comparison of the clinical status and progress of the case was then made. Class A cases were found to be responding favorably to treatment and developing increased resistance. On the other hand, class C cases were doing poorly and failing more or less rapidly. Class B patients, in general, were in fairly good condition. None of these were incipient cases. Red blood-cell counts and hemoglobin estimations on individual patients showed a wide variation among members of the same groups, but the corpuscular volume was found to be much more constant among such members. *Conclusions:* The method as described provided a far better index of the patients' status than hemoglobin values and red-cell counts. The relative blood-cell volume bears a constant relation to the patient's condition. A high value is found in those who are progressing favorably, and a low volume in those who are rapidly deteriorating physically.—*Relative Blood Cell Volume in Pulmonary Tuberculosis, B. H. Schlomovitz, B. E. Hedding and E. J. Kehoe, J. Am. M. Ass., June 7, 1924, lxxxii, 1845.*—(*J. S. W.*)

Reflexes in Pulmonary Tuberculosis.—The lungs are supplied by fibres from both the sympathetic and parasympathetic components of the vegetative systems of nerves. The sympathetics arise from the upper 5 or 6 thoracic segments of the spinal cord. The parasympathetics are represented by fibres of the vagus. All reflexes which take place from the lung must be produced by stimuli which arise in the sensory or afferent fibres of these two systems, and are carried to the cord and medulla, respectively, where they mediate with other neurons to complete the action. The more common reflexes, originating in the lung, which manifest themselves when the pulmonary tissue is inflamed are hoarseness, laryngeal irritation, cough, inhibition of heart, increased muscle tonus and glandular secretion in gastrointestinal tract, flushing of face, spasm of sternocleidomastoid, deviation of tongue from midline, degeneration of facial muscles, flushing of ear, dilatation of pupil, spasm of muscles of shoulder girdle and diaphragm, lessened movement of chest wall partly

due to muscle spasm, pain above second rib and spine of scapula, pain in muscles of shoulder girdle, degeneration of skin and subcutaneous tissue above second rib and spine of the scapula, degeneration of muscles of shoulder girdle. The motor reflex from the lung affects the muscles which are innervated by the cervical segments of the cord. These are the muscles of the shoulder girdle, and the crura and central tendon of the diaphragm. While the reflex spasm is found in practically all the muscles innervated by the cervical segments of the cord, spasm of the following has proved of greatest value in diagnosis: sternomastoids, scaleni, pectoralis major and minor, trapezius, levator anguli scapulae, rhomboidei, diaphragm. In spasm of these muscles in the presence of pulmonary inflammation we are dealing with the same process as in the well-recognized rigidity of abdominal muscles in appendicitis. The impulses are carried upward by intercalated neurons and mediate with the motor neurons arising in the cervical segments of the cord. This shows how the path of reflexes is determined by embryological conditions, for the lungs embryologically are given off from segments anteriorly to the heart, aorta and other thoracic viscera; and consequently are connected with the skeletal structures by neurons which are innervated mostly anteriorly. To determine increased muscle tension, the patient must be in the erect position. The spasm of the muscles is recognized as an increased tension on palpation and is best appreciated when one lung only is involved. When both lungs are involved it is necessary to have in one's mind the feel of a normal muscle as a standard of comparison. Limitation of motion of the chest wall is brought about by spasm of the muscles which fix the sternum, through shortening in the sternocleidomastoid and the first and second ribs, through the scaleni; and below, through the shortening of the crura of the diaphragm. Limitation of motion may also be caused by other factors at times, such as acute pleurisy or pleural adhesions; also by loss of pulmonary elacticity or of pulmonary tissue. It is also commonly observed as an accompaniment of early pulmonary lesions when these latter conditions do not obtain, and when the motor reflexes in the diaphragm and apical muscles seem to afford the only logical explanation. Acute pain is not as important a symptom in pulmonary disease, unless the pleura also is affected, as it is in pleural and peritoneal involvement and inflammation of the abdominal viscera; nevertheless, it is a symptom often found. Receptors for a certain type of sensation are developed only when they are required by the presence of the particular stimulus, and the lung, being a centrally located and protected organ, has never had cause for the development of a delicate pain sense. While acute pain is rarely present as a reflex from the lung, there is a certain amount of irritability and uncomfortable sensation, also changes in touch, heat, and cold perception, which are transmitted to the surface of the body over the zones supplied by the cervical nerves. These areas consist of the tissues covering the neck and upper portion of the chest, above the second rib anteriorly and those lying between the occiput and spine of the scapula posteriorly. There is also a soreness felt, particularly upon pressure or increased by pressure, if otherwise present, in the deeper tissues. Soreness over the pectoralis and trapezius is often complained of by tuberculous patients, whether with or without pressure. Pleural pain may be expressed in any of the intercostal nerves. As a rule it is confined fairly closely to the areas over the point of inflammation, except when the costal portion of the diaphragm is involved. Consequently, most pleural pain is expressed over the surface of the chest from the clavicle above to the costal border below. In case of pleural inflammation involving the costal portion of the diaphragm, however, the pain very often extends down over the abdomen. Pleural pain may not only be superficial but may also be expressed in the sensory nerves which supply the deeper structures. The trophic reflex from the lung is expressed in tissues supplied by those neurons which receive and transmit stimuli from the lung to the superficial structures of the body. These trophic reflexes may be evident on the surface of the body. Thus, one can often see a thinning out of the skin and subcutaneous tissue between the second rib and the angle of the jaw anteriorly, and between the spinous processes; of muscles which present this atrophy best under the fingers and eye of the clinician are the sternocleidomastoid, pectoralis, trapezius, and levator anguli scapulae. The disease in the skin and subcutaneous tissue shows as a thinning out, a lessening in bulk. In the long and flat muscles it also shows as a change in solidity and rotundity. The muscle, instead of being one solid mass, often becomes a series of bundles. All the tissues show a loss of elasticity—a feeling of doughiness—upon palpation. After one has grasped the neurology which underlies these changes, and attained skill in detecting them, he has at a glance very important suggestions as to the presence or absence of past or present chronic inflammation in the lung.—*The Importance of a Knowledge of Reflexes in the Diagnosis of Pulmonary Tuberculosis*,

F. M. Pottenger, Med. Jour. & Rec., February 20, 1924, cxix, 181.—(B. T. McM.)

Pulmonary Tuberculosis and Vegetative Nervous System.—Testing the reactivity of the vegetative nervous system makes possible proper treatment, especially in regard to the value of various specific and nonspecific methods. The coincident use of two opposing irritants, adrenalin and tuberculin, results, in individual cases, in removing pyrexia (only 3 of 18 cases were thus permanently influenced) and probably also occasions increased resistance in nonfebrile but tuberculin-sensitive individuals. The use of calcium in tuberculosis therapy could be further restricted. Prolonged oral use of daily amounts of 4 to 5 gm. calcium chloride appears to inhibit exudate formation during artificial pneumothorax. In combination with digitalis, which reinforces its action, individual cases of pneumonic-exudative tuberculosis quickly became afebrile. An influence upon the process itself in these cases is not unlikely. Continued use of atropine, alone or combined with other methods, gives similar results.—*Lungentuberkulose und vegetatives Nervensystem. IV. Mitt. Therapeutische Folgerungen, E. Guth, Beitr. z. Klin. d. Tuberk., April, 1923, lv, 41.—(H. J. C.)*

Pulmonary Tuberculosis and Vegetative Nervous System.—From the large amount of literature available it appears that in proliferative tuberculosis the excitability of the sympathetic nervous system is practically normal, but in progressive, exudative tuberculosis it is definitely reduced. Whether or not tuberculosis is more likely to develop in a sympatheticus-weak than in a sympatheticus-strong individual is, however, difficult to decide. On the assumption that weakness of the sympathetic indicates a poor prognosis, measures to raise sympathetic tone are indicated. Adrenalin is useless, as has been repeatedly demonstrated in Addison's disease. Numerous results, however, suggest an intimate relation between certain electrolytes and muscular and nervous tone. Particularly is this true of calcium. There is good reason for believing that calcium therapy is valuable in tuberculosis in raising sympathetic tone.—*Beziehungen Zwischen Lungentuberkulose und vegetativem Nervensystem, K. Kading, München. med. Wchnschr., February 22, 1924, 225.—(E. R. L.)*

Vital-Capacity Standards for Women.—It has been shown by numerous and ample studies that there is a definite relation between the vital capacity of the lungs and the amount and type of pathological conditions within the chest and their course and effect on the patients' activities. The most important thing is to determine and unite on satisfactory standards. The present observations were made on a group of 738 college women at the University of Minnesota varying in age from 16 to 44. The measurements excepting the vital-capacity determinations were taken by the department of physical education during routine physical examination. The vital capacity was determined by a Sanborn water spirometer, the highest of three trials being recorded. Using Dreyer's formula calculated from body weight $\frac{\text{W. O. } 72}{\text{V. C.}} = K$, appoximately 77 per cent were up to 90 per cent or more of the normal standard. Using Dubois's formula calculated from the surface area, approximately 64 per cent were up to 90 per cent or more. Using a table of accepted standard weight formulas prepared by Meers, only approximately 41 per cent were up to 90 per cent or more. The correlation between stem height and vital capacity was found no better. If we assume that the normal standard should be 100 per cent and the normal deviation above or below not more than 10 per cent, the present standards for women would seem to be too high, especially those calculated on a basis of height. The highest average vital capacity was found at the age of 23, namely 90 per cent.—*A Comparison of Normal Standards for the Vital Capacity of the Lungs of Women, R. E. Boynton, Arch. Int. Med., March 15, 1923, xxxiii, 292.—(A. P.)*

Effect of Past Disease on Vital Capacity.—Practically all investigations have found a considerable percentage of unexplained low readings in supposedly normal persons. This study was undertaken to determine the effect of certain past intrathoracic diseases on the vital capacity. For this the Student's Health Service Records of 1,517 male students comprising the freshmen class entering the University of Minnesota in 1922 were available. Vital-capacity readings were taken with a Sanborn water spirometer. Students giving histories of epidemic influenza, pneumonia, pulmonary tuberculosis, or heart disease, or showing physical signs of the three latter, were studied specially. A curve was prepared for each disease showing the normal distribution of vital capacities, with that for the particular group studied superimposed, the areas between being shaded to indicate shifting, either to the left (subnormal) or to the right (supernormal). There were 611 students in these combined special

groups. The normal vital capacities were computed by averaging four determinations: weight, sitting height, surface areas, and chest circumference. *Influenza:* There were 333 students with a history of influenza (only cases occurring between 1917 and 1920 were considered). There was a slight lowering and shifting to the left of the curve, indicative of a slight reduction in vital capacity of the group as a whole. *Pneumonia:* There were 189 students with a history of pneumonia. A similar but slightly more marked lowering of the curve and shifting to the left was noted. *Pleurisy:* There were 55 students who gave definite histories of severe or repeated attacks, or showed physical signs of pleurisy. There is a marked lowering of the height of the curve and a shifting to the left, the shaded area being considerably increased. *Tuberculosis:* Only 20 definite cases, present or past, were found. A marked reduction in lung capacity of the group as a whole is evident. The same was true of 14 cardiac cases although apparently compensated at the time of examination.—*The Effect of Certain Past Diseases on Vital Capacity, W. P. Shepard, Arch. Int. Med., February 15, 1924, xxxiii, 185.—(A. P.)*

Output of Urea at High Altitudes.—Physicians in the high Alps have long been familiar with the unstable equilibrium which characterizes the acclimatization period in cases of pulmonary tuberculosis. Focal reactions and initial loss of weight are common examples of this tendency. To determine whether an enhanced metabolism would show itself by an increased excretion of urea during the first months after arrival, urea estimations were done on the urines of five patients. The hypobromite method was used throughout and the specimens were taken at weekly intervals beginning two days after arrival. The nitrogenous intake was not controlled. All cases showed marked irregularity in the twenty-four hour output of urea from week to week for the first two months following their arrival.—*A Note on Variation of Output of Urea During Acclimatization to High Altitudes, A. F. Bull and F. H. Healy, Tubercle, July, 1924, v, 490.—(J. S. W.)*

Water Economy of Body in Pulmonary Tuberculosis.—Tuberculin calls forth characteristic changes in the disturbed water-economy relations in pulmonary tuberculosis which are, however, not of specific nature, since nonspecific medicaments and hygienic procedures (pyramidon, fresh air treatments, and to a certain extent pneumothorax treatment) are also capable of producing these changes. They are also not only characteristic for tuberculosis since they occur in nontuberculous conditions (echinococcus and apoplexy). The "water reactions," of Meyer-Bisch, of tuberculous individuals are not limited to the three described types; the number is dependent more upon the possible combinations of weight and serum concentrations with unchanged, increase or decrease of both functions of the water economy. Prognostic conclusions of far-reaching significance are not possible on the basis of the behavior of the water economy. The value of therapeutic measures cannot be judged more from the behavior of the water economy than from the clinical course of the case, since the factors of the water economy are too numerous and too difficult to control. As clinical methods the water-economy determinations are too complicated and unsuitable, but they may serve to give insight into the morbid physiology of the diseased body. The determination of the specific urine weight-curve has also proven a noteworthy factor, like serum concentration.—*Wasserhaushalt bei Lungentuberkulose und seine Beinflussung durch spezifische und unspezifische Massnahmen, E. Seuffer, Beitr. z. Klin. d. Tuberk., April, 1923, lv, 1.—(H. J. C.)*

Right Upper Lobitis in Pulmonary Tuberculosis.—Attention is drawn to a peculiar and interesting localized form of the disease limited below by the superior interlobar fissure of the right lung. It is shown clearly by physical signs, as well as by radioscopy and radiography. Five cases in point are cited. The usual radiographic picture is that of a dense homogeneous shadow, in the midst of which is commonly seen cavity formation, giving a bread-crumb or honey-comb appearance, or occasionally a large area of rarefaction. What especially characterizes the picture is the clearness of contour and the well defined, often linear lower border. The stethoscopic signs commonly obtained are dulness, exaggerated fremitus, and more or less tubular or cavernous breathing. Râles are generally heard after cough, usually either fine or bubbling in character. Pleural friction sounds are not rare. This type of lesion is seen only in the chronic, slowly progressing forms of tuberculosis, and there is usually evident some tendency to fibrosis. A similar picture in the opposite lung is most unusual. This lobitis must be differentiated from a localized pleurisy, or interlobitis, which may at first glance simulate it. It is interesting to speculate on the pathogenesis of this sort of lesion. It is believed that such

patients usually have a history of illness beginning a long time back, the initial symptoms often having escaped observation. It is believed that apical lesions generally represent the end of a primary infection dating from childhood. When later a reactivation occurs and they progress, a fissural reaction limits their progress. This type of lesion responds very favorably to pneumothorax treatment, which is usually indicated. While adhesions might be expected, as a matter of fact they rarely interfere with collapse. When there is a pleural symphysis, either Tuffier's operation or thoracoplasty would seem indicated. —*La lobite supérieure droite localisation de tuberculose pulmonaire, L. Bernard and Beythoux, Rev. d. l. Tuberc., September, 1923, iv, 471.*—(*A. P.*)

Ghon's Primary Complex Viewed by X-Ray.—Ghon's primary complex can also be determined by means of the roentgen rays. It is more frequent in the right than in the left lung and more so in the right lower fields. The majority of the cases studied (78 per cent) revealed a single focus in the lung. Density and limitation of the shadow serves to differentiate fresh and older foci. Clinically the tuberculous primary complex is frequently symptomless; there is a marked tendency to healing and therefore it requires little in the way of treatment and especially no specific treatment.—*Über den tuberkulösen Primärkomplex in der Lung mit besonderer Berücksichtigung des Röntgenbildes, E. v. Müller and E. Klinckmann, Beitr. z. Klin. d. Tuberk., May, 1923, lv, 133.*—(*H. J. C.*)

X-Ray Observations on Pulmonary Tuberculosis.—The greatest knowledge of the pathogenesis and pathology of pulmonary tuberculosis has come from two sources—postmortem studies and animal experimentation. Armed with this knowledge it is possible for the trained observer to lay bare the living lungs on the X-ray plate and tell us what has happened, what is happening and what probably will happen. The X-ray is the study of anatomy in the living. An outline of the pathogenesis and progress of tuberculosis is given. The first or primary injection of live tubercle bacilli in an animal results in a slight lesion at the point of inoculation but with a marked reaction of the regional lymph nodes. When the animal is again inoculated Koch's phenomenon results, characterized by a vigorous reaction at the point of reinoculation. In studying the pathology of pulmonary tuberculosis we must remember that this can mean anything from a small nodule with its characteristic cell structure to a whole lung which has become solid or a lobe entirely honeycombed with cavities. The anatomic factors of defense present in the respiratory tract are the ciliated epithelium and the lymph vessels and lymphoid tissue. The pleura has an extremely rich supply of lymph vessels. But beyond the end of the *ductulus alveolaris* there are no lymph vessels, no lymph flow and no lymphoid tissue. Here the phagocyte is the defender against invasion, carrying dirt and bacteria within the realm of lymphatic defense. The normal lymph flow in the lung is from the parenchyma toward the hilum, except for a narrow strip of lung tissue immediately under the pleura which can drain into the pleura. The flow in the pleura is over the surface of the lung to the lymph nodes at the hilum. The direction of the flow is established and guarded by valves and is maintained chiefly by the massage of the vessels due to the repeated movements of respiration plus the inherent elasticity of lung tissue. The apices undergo a very limited movement; hence their lymph flow is sluggish and the lodgment of invading tubercle bacilli is comparatively little disturbed. On the other hand, at the bases, where the lymph flow is free, the X-ray shows that this is usually the last part of the lung to become infiltrated by tubercle. In the vast majority of cases of pulmonary tuberculosis, the bacillus enters the lungs by inhalation. It finally reaches the divisions of the primary lobules where it is taken up by the large phagocytes and carried to the lymphoid tissues. Here destruction of the bacillus is accomplished or pulmonary tuberculosis begins. With the implantation of tubercle in the tissues of the body the pathological changes which immediately result vary with every individual. But most striking are the differences between adult and child pulmonary tuberculosis chiefly because there are examples of reactions in a sensitized and a nonsensitized animal. Pulmonary tuberculosis of early childhood does not affect one part of the lung oftener than another. In the first year of life there is almost no tendency for the lesion to heal but progressive caseation occurs; cavities are uncommon but do occur; dissemination readily takes place and miliary tuberculosis is a frequent termination. The lymph nodes at the hilum are invariably markedly involved and usually are the earliest and chief centre of the disease. The true primary focus (Ghon) is not in these nodes, however, but in the lung parenchyma; such hilum involvement has all the characteristics of first infection. With increasing age, however, there is a marked tendency for the

primary stage to heal. The early puerile lesion, as seen on an X-ray plate, consists of a small circumscribed area of density connected to the hilum by a heavy trunk, usually seen within the midlung zone, but occurring throughout all lobes, only rarely at the apices. The hilum shadows are heavy, showing densities suggestive of calcification, caseation, or fibrosis. Sometimes enlarged pulmonary or bronchial nodes may be seen. Thickened trunks and enlarged hilum shadows on an X-ray plate may be caused by various other childhoood diseases, such as measles, whooping cough, or mumps. They can never be considered as being the result of tuberculosis unless calcification or caseation is definitely shown to be present. Adult apical tuberculosis resembles in all respects the reaction to the second infection in experimental animals. It is the reaction of an infection in a sensitized animal. Active pulmonary tuberculosis in the adult is usually the result of repeated infections. The focal lesions of early life which have healed are beneficial but they do not always effectively protect. The patient has relative immunity but a diminution of resistance is brought about by intercurrent disease, fatigue, malnutrition, and various other factors. Immunity diminishes when recovery occurs and disappears after the lesion is completely healed, but is increased by reinfection spread or metastasis of the active disease. Before a child is ten years of age he is usually sensitized and has acquired a degree of immunity. Infection after this age produces the picture of adult tuberculosis with apical localization, tendency to chronicity with fibrosis, cavity formation, and noticeable absence of associated tuberculosis in the regional lymph nodes; in brief, a local reaction in an allergic soil. This reaction is characterized by the formation of an exudate at the point of inoculation which is the most striking difference between adult and puerile types of tuberculosis. Peculiarly, the exudation takes place immediately under the pleura. This has been confirmed by X-ray observations. The exudate may be at first of the polymorphonuclear type but is rapidly replaced by a mononuclear exudate and proliferation of fixed tissue cells. From these elements the characteristic tubercle is formed from which point the pathological changes progress either to the reparative changes of fibrosis and calcification or the destructive changes of caseation, ulceration and cavitation. These pathological changes, as shown on an X-ray plate, vary in their quality and degree of density, progressing from the least dense to the heaviest, as follows: serous exudate, cellular exudate; fibrosis; caseation, calcification. After the apical lesion, the most common lesion of adult pulmonary tuberculosis is caseous bronchopneumonia. It is invariably associated with old apical lesions, usually containing cavities. It is probably caused by the aspiration of bacilli-infected sputum. Upon the X-ray plate, it is easily recognized as a diffuse heavy mottling frequently seen scattered along the main trunks suggesting "raisins upon a stem." It may become confluent and produce pseudolobar caseous pneumonia. Recent studies have shown that "caseous tubercle can heal by resolution, and finally disappear completely." Acute miliary tuberculosis is believed to be the result of lack or loss of immunity and is the result of reinfection from older foci. The X-ray picture is that of fine, sharp, discrete studdings more or less evenly distributed throughout all lobes. The X-ray diagnosis is justified only when the subject gives no history of work in a dusty trade, especially anthracite coal mining. Many cases of healed miliary tuberculosis are now being found by the X-ray and, most peculiarly, these patients seldom, if ever, give any history suggesting serious lung infection. In conclusion, the authors strongly question the conception that there is first hilum tuberculosis (bronchial nodes) and then peribronchial tuberculosis and finally parenchymal tuberculosis. They believe the infection enters the lung by the air passages or the blood-stream and is carried from either to the lymphoid tissue which is directly connected with the lymph vessels. In the child there is little reaction at the point of inoculation, but a great reaction of the regional lymph nodes. In the adult there is comparatively little reaction of the lymph nodes, but massive reaction around the infection, with extensive exudate, which floods a definite part of the lung. The only tuberculous lung lesion, observed on the series of X-ray plates of a given patient, that spread from the hilum to the periphery was tuberculous caseous bronchopneumonia. It can originate from the hilum only when a tuberculous lymph node ruptures into a bronchus, and infectious material is aspirated into the smaller air passages.—*X-ray Observations of the Pathogenesis of Pulmonary Tuberculosis, H. K. Dunham and J. H. Skavlem, Tubercle, February, 1924, v, 217.—(J. S. W.)*

Annular Shadows in Pulmonary Tuberculosis.—The use of the term *annular shadow* has led to a great deal of confusion, and, until its significance is settled, promises to interfere seriously with the proper therapy of lung cases which show these shadows. Those who contend that the shadows indicate localized pneumothora-

ces disagree as to the frequency of incidence, Barlow and Thompson stating that pneumothorax is the rule rather than the exception in lung tuberculosis; but among the author's 800 cases at the Arequipa Sanatorium, the number of distinct spontaneous pneumothoraces does not exceed 3 per cent. Autopsy evidence is lacking to support the pneumothorax theory, and the pathological explanation of the process as a pleuritic phenomenon as given by Honeij and by Amberson is against known evidence, and seems made to fit a theory. A number of observers have argued that cavities do not disappear as rapidly as pleural shadows, but, on the contrary, cavities as large in diameter as 3 or 4 even 5 cm. can close in a relatively few months if conditions are favorable. A cavity may assume all the characteristics which have been ascribed by Honeij to localized pneumothorax. To detect the softening and excavation of tuberculous deposits in the lungs, the increase or appearance of râles after cough following expiration is one of the most dependable signs when weighed with impaired resonance, but it may be so simulated by pleural sounds as to make differential diagnosis dependent on the best of X-ray plates. The reasons for believing that the interpretation of certain, or any, annular shadows as localized pneumothoraces is wrong are these: *1:* Many cases of spontaneous pneumothorax were observed but none were round; the same is true of small artificial pneumothorax pockets. *2:* Anteroposterior and lateral views of these conditions show them to be equally round in both directions, which could be true only if they were cavities. *3:* Artificial pneumothorax will first compress and then collapse these shadows and move them from their original positions, which would be impossible if they were in the parietal pleura. *4:* If unilateral, and found sometimes to contain fluid, the shadow may be studied by X-ray in the morning after the patient has spent the night on the affected side, before the cavity has emptied itself and after postural effort at emptying. Pneumothoraces containing fluid will not empty. *5:* Stereograms, taken in one-sixtieth second each, show in great detail that these shadows are cavities, orient them in their relation to the chest-wall, show their connection with bronchi, and how they increase in size by coalescing with adjacent and often very small cavities or by including softened areas which border on them. All annular shadows should be considered as cavities until diligent investigation proves that they are to be better interpreted some other way.—*Annular Shadows: Are They Cavities or Spontaneous Peneumothoraces, P. K. Brown, Am. J. Roentgenol. & Rad. Therap., June, 1923, x, 445.—(J. B. A.)*

Annular Shadows in Pulmonary Tuberculosis.—In a boy aged 16, in whom there were signs and symptoms of active tuberculosis of hilar origin in the right lung, a large annular shadow appeared in the right side in the X-ray picture, where, a month previously, no such density existed. Over its site there were no signs of cavitation, but there was a pleural rub at or near it. The shadow increased and active symptoms persisted; at the end of twenty-five days artificial pneumothorax was induced on this side. X-ray reproductions accompany the article to show the changes which occurred thereafter. After the third refill the lung was well collapsed, except where it was in contact with the anterior chest wall at the site of the annular shadow. Later the adhesion here gave way, a better collapse was obtained, and the shadow became much reduced in size and less distinct. The shadow was diagnosed as due to some change in or on the visceral pleura because it occurred about the same time as an attack of pleurisy, it coincided in position with a pleural adhesion, it appeared to be on the surface of the lung, not inside it, and its remains were visible on the surface of the collapsed lung. It was not cavity because it formed too rapidly, it did not show decreased density in the centre, it did not appear to be in the substance of the lung and was hardly visible in oblique position, it gave no stethoscopic signs of cavitation, and it retained its circular outline, instead of falling in as the lung collapsed. As a rule tuberculous cavities form by the coalescence of small cavities in solid lung. Large cavities are spherical only if they are of long standing. Cavities may be obliterated in time, but the process is a slow one and depends upon dense fibrotic contraction of the surrounding lung. Large annular shadows are probably due to pleural changes and have no important clinical significance, especially if they appear quickly, change in size, show no increased translucency in the centre, or finally disappear. On the other hand many of the smaller annular shadows, even though they appear in a comparatively healthy area of lung, are caused by tuberculous cavities.—*Annular Shadows in Pulmonary Tuberculosis, G. T. Hebert, Tubercle, February, 1924, v, 212.—(J. B. A.)*

Roentgenology in Tuberculosis with Diagnostic Criteria of Cavities and Pleural Annul r Shadows.—For the purpose

of X-ray study pulmonary tuberculosis is divided into four main types: miliary, chronic ulcerative, chronic fibroid, and glandular. In miliary tuberculosis involving the lungs the radiogram shows numerous minute areas of radioöpacity, varying in size and shape, and of a somewhat irregular outline scattered between the radiolucent linear markings of the bronchial tree, evenly distributed throughout the fields, sometimes reaching the periphery; the pleura, being inflamed and congested, imparts a peculiar blurring to the picture. Chronic ulcerative tuberculosis casts shadows of various sizes and densities; if the process is recent the shadows are soft and blurred, and if of long standing they are denser, more rounded and clean-cut. Repair may follow with connective tissue casting its shadows in the picture, or a rarefaction due to cavitation may appear. For diagnotic purposes cavitation may be regarded as acute or chronic. The following facts must be considered in studying acute cavities: (1) they are improbable in the absence of an extensive involvement; (2) their shape is triangular or quasitriangular; (3) their outline must be irregular; and (4) they show no evidence of capsule. Acute cavitation is not to be expected unless the infection is well established. Following the anatomic structure of the lnug such cavities assume a pyramidal shape, their internal borders being ragged and irregular because of the asymmetry of the destructive process. Being situated usually fairly deep in the parenchyma, normal lung markings can be seen crossing the area of rarefaction. Chronic cavities have a distinct fibroid wall. At first they tend to maintain the triangular shape, but later, due to the surrounding induration and shrinkage, they become circular. The capsule is represented on the radiogram by a dense ring-like shadow of varying thickness, having either a well defined or a blurred but rough, ragged, uneven internal border enveloping a radiolucent area; this may or may not contain lung markings. Four different types of shadows are to be dealt with in the differential diagnosis of tuberculous parenchymal cavitation: (1) those pertaining to true cavities, (2) those cast by thickened pleura in annular formation, (3) those cast by localized spontaneous pneumothorax; and (4) those cast by annular shadows of fibroid origin. Cavities are found most commonly in the upper lobes, usually just below the clavicle close to the periphery, sometimes at the apex, and seldom at the lower portion of the upper lobe. Pleural shadows may occur anywhere in the field, most commonly posteriorly at or near the central portion. Occasionally, when they are of interlobar origin, they are visualized in the midst of the lung parenchyma; at times they are seen directly behind the front ribs. The type of spontaneous pneumothorax that may be mistaken for cavity is always situated at the apex, unless it accompanies the pleural annular shadows, with cupping of the encircled lung tissue, when it may occur anywhere, but usually in the central portion of the field close to the posterior surface. Fibroid changes in circular formation are invariably seen originating from the hilar region and are, therefore, in the centre of the field, and may travel either fowards or backward, sometimes upward and rarely downward. The size of a pulmonary excavation varies, but is always in direct proportion to the nature and extension of the lesion. In those cases following a caseating course from the beginning, cavities usually attain enormous sizes, while in those cases in which fibroid proliferation starts early, cavities are small and commonly multiple. Annular shadows of pleural origin may be of any size, regardless of the degree of involvement. Pleural shadows have been seen that were the only unmistakable evidence of the true nature of the accompanying constitutional condition, the remainder of the field being comparatively clear, showing only scattered shadows to awaken suspicion concerning their real cause. Fibroid shadows in annular formation are to be expected, and are met with as a rule in cases of true fibroid phthisis, although, at times, they are seen in the ulcerative type when fibroid changes follow, step by step, the parenchymal infiltration. They are usually small and are seen connected by a stem to the hilar region whence they originate. Spontaneous pneumothoraces of apical origin usually involve the whole of the apex. When associated with pleuritic annular shadows, their size varies considerably. Acute cavities, being deprived of a capsule, or, at least, of a complete capsule, when situated at the very apex, cast a shadow that may be mistaken only with the shadow cast by a spontaneous pneumothorax; in the latter, however, the edge of the collapsed lung can often be made out, and the shadow is one of radioparency containing no lung markings. When acute cavities occur elsewhere, they represent a "punched-out" area of radiolucency showing lung markings, of triangular or almost triangular shape, and of irregular, ragged, ribbed outline. No other pathological entity casts a similar shadow. Chronic cavities, however, no matter where situated, must be differentiated from annular shadows and fibroid ring-like shadows. Annular pleural shadows have a clean-cut, even, smooth internal border, much different from the

external one, which has a blurred and undefined appearance, the dense shadow gradually fading out as it travels outward away from its axis. On several occasions the author has diagnosed triangular shadows showing the above characteristics as of pleural origin, autopsy findings confirming the belief. One could agree with the recent statement that "pneumothoraces are never annular in contour," if one may add, "when accompanied by pleural changes." Hard knocks and disappointments have taught us to believe in the existence of annular areas of thickened pleura with subsequent cupping of the encircled lung parenchyma. Autopsy findings have placed our error in the limelight by revealing annular pleural changes where findings of a cavity had been foretold because of the leading physical signs and because of what we considered positive X-ray findings. It was true that in the process of separating the adhesions between the parietal and visceral pleura much of the structural appearance was destroyed, but there was often sufficient pathological evidence at hand to prove the existing conditions. On inflation of the lung, a well-pronounced umbilication of the parenchyma was observed at the site of the lesion before inflation was completed, this portion being the last to attain full expansion. After the lungs were properly expanded they were submerged in water, and in only one case there seemed to be a few air bubbles that did not come up to the water surface, showing that if there were any air leakage it was almost osmotic in character. This naturally might have been the result of traumatism during dissection. The post-mortem work performed by the author has failed to confirm the theory of ruptured lung as causative of pneumothoraces simulating cavities. Every case of ruptured lung that has come under observation has been accompanied by the extensive pneumothorax, with partial or complete collapse of all lobes of the affected side. When the diagnosis of a peculiar shadow is in doubt fluoroscopy is used. When a shadow is being cast by a cavity, it is obvious that the excursion of the chest-wall in the neighborhood of the area being studied is considerably impaired, but not altogether absent; in true pneumothorax, there being a collapsed portion of the lung, chest movements are absent, while in cases of annular pleura, there is a peculiar lagging at the involved zone which interferes somewhat with the normal expansion. When a shadow is being cast by fibroid changes, there is no appreciable change in the thoracic movement. Autopsy evidence leads to the support of Amberson's hypothesis, "that in a large majority of instances, so called roentgenographic pleural annular shadows accompanying pulmonary tuberculosis do not represent localized pneumothoraces secondary to lung rupture, but indicate rather the presence of simple chronic or subacute localized pleurisy with or without localized retraction and separation of the pleural layers." It is also believed "that these shadows develop and enlarge as accompaniments of intrapulmonary tuberculosis that is usually active or progressive." Equally, Honeij is endorsed in his statement that pleural shadows have no relation to the extent of a pulmonary infiltration or softening and breaking down of tissue, as this is in accord with the contention that cavities are not to be expected in the absence of a well established tuberculous involvement. Judging from personal observation, Ramos-Casellas cannot endorse the teachings recently put forward to "consider all annular shadows as cavities until diligent investigation proves that they are to be better interpreted some other way." Why not consider them as of pleural origin in the absence of leading roentgen and physical signs of cavity? It has been proved by serial roentgenograms that annular pleural shadows behave very differently from those of cavities. In one case, a well-developed pleural shadow which had revealed itself in serial stereograms for nearly a year disappeared in three weeks. Cavities once developed are there to stay, even though their size and shape may be altered during the process of repair. The presence of fluid within an annular shadow constitutes no pathognomonic sign of pulmonary cavity. The author, like others, has drawn fluid from localized pleurisies in annular formation and has seen the pleuritic process disappear in a comparatively short time. Artificial pneumothorax helps considerably in the diagnosis of doubtful cases of cavitation. In the presence of induced pneumothoraces, annular shadows of pleuritic origin either disappear or else, when they are of long duration, and extensive adhesions have formed binding the parietal and the visceral pleuras, adhesive bands are seen strung from the visceral to the parietal layer crossing the pneumothoracic field. Cavities, on the other hand, change in position following the collapsed lung in its displacement and become distorted in appearance. When acute, they may so obliterate as to be scarcely discernible. Fibroid tuberculosis is shown upon the film by linear markings in reticular formation encircling, here and there, irregular areas of increased density, or by dense ribbon-like linear markings radiating from the hilum to the periphery. In hilum tuberculosis there is an almost pathognomonic

uneveness of the density of the shadow, having undefined areas of radioparency intermingled with areas of radioöpacity. The sharp well-defined margins of a normal hilum are lacking, the shadow fading gradually from its centre so that its borders are blurred and undefined. Traveling in all directions into the parenchyma, which has a normal appearance, short, heavy, blurred trunks are seen originating from the margins of the hilum and losing their density from within outward. In the majority of cases, in a short period of time, the disease becomes parenchymatous. In children hilum densities are not so significant. The X-ray characteristics of tuberculous pleurisy and empyema are considered.—*Roentgenological Synopsis of Pulmonary Tuberculosis with Special Reference to the Diagnosis of Cavitation, P. Ramos-Casellas, Southwestern Med., March, 1924.*—(*J. B. A.*)

Autopsy Report on Roentgenographic Pleural Annular Shadow.—A review of certain French work on peculiar pleural lesions in pulmonary tuberculosis, and especially of the publication of American authors on the subject of annular shadows is given. As to the lesion casting these shadows, there are divergent views; some believe it is a simple localized pleurisy, some that it is a localized pneumothorax, and some that all annular shadows represent intrapulmonary cavities. A typical case which was followed to a fatal termination is reported. The patient, a woman, aged 37, entered the Ivry Hospital, May 13, 1922, giving a history of symptoms of tuberculosis starting in the summer of 1921. In January, 1922, she entered the Cochin Hospital and remained there for three months, during which she had slight fever, night sweats, cough and expectoration with tubercle bacilli. During her stay at Cochin, three attempts to induce artificial pneumothorax on the left side were made but were unsuccessful because of the presence of pleural adhesions. On admission to the Ivry Hospital the general symptoms were only moderate, the fever being slight, and the bacilliferous expectoration was not abundant. Physical examination of the lungs showed harsh expiration on the right, anteriorly, with crepitant râles after cough posteriorly; on the left, dulness anteriorly, and fine râles after cough. In the lower two-thirds of the left lung there was an area of fine crepitant râles mingled with friction sounds, resembling those heard on certain corticopleuritides. Radioscopy showed on the left toward the axilla an unexpected annular shadow, appearing like a shell of 2 or 3 mm. thickness and 6 cm. in diameter, and surmounting a zone of total opacity at the base where the heart and diaphragm shadows were confused. The "shell" enclosing the space mentioned could be made out as the chest was viewed in different planes. Coughing by the patient produced no change in the volume of the shadow. The image was displaced *en bloc* by the movements of expiration and inspiration. The appearance was that of a cavity but it seemed difficult to admit that it could be completely overlooked on examination, and, in the absence of physical signs of cavity, the presence, at the base, of suppressed breath-sounds and of friction râles, and, in view of the functional and general symptoms which the patient presented, a provisional diagnosis of a deep corticopleuritis of the left lung was made. During the summer of 1922 the patient's temperature rose at times to 39°C. but subsequently the symptoms improved and for some months the temperature did not go above 38°. During this period of amelioration the physical signs became accentuated. The friction sounds persisted at the left base and in October, 1922, a "tubopleural" murmur was heard in the middle zone of the left posteriorly and in the axilla. A new radiograph showed the persistence of the annular shadow but the lung seemed compressed toward the summit. In February, 1923, a tubocavernous murmur was heard in the upper third of the left lung, while in the lower two-thirds the râles persisted as previously. In May, 1923, fever became more marked, dyspnea appeared, the general condition was worse, and there were signs of a caseous process in the right lung. At the same time there were signs of an enormous cavity on the left; this was shown by a radiograph made about three months before death, the cavity being limited by a thick annular border. The patient died September 11, 1923. At autopsy the right lung showed tuberculous bronchopneumonia without cavitation. There was a scar at the apex, and pleural thickening becoming very marked over the diaphragm; there was subpleural caseation in the interlobar fissures which were obliterated. Dense adhesions also extended to the mediastinum and pericardium. On the left there were firm adhesions and a very marked thickening of the diaphragmatic pleura. At certain points the pleura, which was almost 2 cm. thick, was almost cartilaginous. Superficially the direction of the fissure could not be recognized. On section an enormous cavity occupying four-fifths of the upper lobe was found; this was definitely limited internally by a band of pleural thickening representing the interlobar fissure, because one could see it continued at

the base with thickening of the diaphragmatic pleura which was almost cartilaginous. The parts which remained of the lung (lower lobe) presented cavity alterations or advanced bronchopneumonia. Microscopic tissue sections showed the expected changes. This case seems at first to support certain points of view of those authors who would assume that this annular shadow represented a true intrapulmonary cavity. But a close study of the clinical history compared with the autopsy specimen and the radiographic findings indicates that Amberson is correct when he assumes that these annular shadows are due to thickening of the pleura and that they are independent of the evolution of the lesions of the subjacent lung. The symptoms and the clinical progress were those of a combination of pulmonary and pleural lesions designated corticopleuritides by deJong and Bezançon. These deep corticopleuritides may accompany lesions progressing toward the interior of the parenchyma, and can give rise to a syndrome of cavity at the base, or they may become stationary and remain for long periods of time without affecting greatly the general condition. In this case the fact that the cough did not modify the annular shadow, which however moved with the rest of the lung during the respiration, is emphasized in connection with other findings. The pleural lesion found at autopsy was not of the type commonly seen in tuberculosis, but constituted an important pleuritic process which was probably caseous at some time, later becoming transformed into the thick cartilaginous formation. After a year, when the patient's condition suddenly grew worse, the process of caseation returned with increasing intensity, destroying the lung contained in the thickened pleural shell, to terminate in the formation of the enormous cavity. The adhesions present on the surface of the lung helped cause the deviation of the fissure from its normal course, thus giving rise to semicircular or triangular shadows. The mechanism of the process is explained as follows: Following the evolution of the lesion of the subjacent lung, either the shadow persists, then fades when the case progresses favorably, or it may increase and correspond at a given moment to cavitary signs, not because it was a question of cavity, silent on auscultation, but because there was produced a caseation of the lung within the zone limited by the annular shadow. This progressive caseation develops into cavity, but subsequently to a process of pachypleuritis which is the true cause of the annular density.—*À propos du diagnostic des cavernes pulmonaires. Un cas "d'ombre annulaire radiographique" des auteurs Americains avec autopsie, S. I. de Jong and Pestel, Bull. et mem. Soc. méd. d. Hôp. d. Par., March 21, 1924, xlviii, 1.*—(*J. B. A.*)

Radiological Disappearance of Pulmonary Cavities.—Eleven radiographs of 5 cases of pulmonary tuberculosis are reproduced with this article to demonstrate the disappearance, radiographically, of annular shadows interpreted as indicating cavitation. Diagnosis of cavity was made not only through radiographic appearance, but also through physical signs. The authors believe that in the present state of our knowledge every radiographic image with the appearance of cavity in the pulmonary field in a case of tuberculosis indicates a cavity, unless there be evidence to the contrary. Two mechanisms may be invoked to explain this disappearance: sclerosis of tissue with retraction and masking by emphysema developing round about the antrum.—*La disparition radiologique des cavernes pulmonaires (4 planches hors texte), C, A. Piguet and A. Giraud, Ann. d. Méd., Par., November, 1923, xiv, 363.*—(*W. B. S.*)

Mechanism of Larynx.—Although it is generally stated that the larynx is the organ of voice, on examining this organ in various types of animals one is impressed by the fact that there is no marked relation between the structure of the organ and the vocal powers of its possessor. Vocal cords, similar to those of man, are to be seen in many animals which are naturally silent, as in most bears, while there is a complete absence of vocal cords, as we usually picture them, in many habitually noisy species, such as the domestic cow. The primitive lung first makes its appearance as an outgrowth developing from the ventral wall of the foregut, and at its orifice a sphincter band of muscle is developed to keep it closed at all times except when air is passing through; food and water are thereby prevented from entering. A primitive larynx thus comes into existence, with the primitive function of closing the air tract. In the course of its evolution the larynx later takes up a certain position in order to subserve functions of olfaction and respiration, and the peculiar situation it occupies in man can be understood if various structural characteristics of the human being are taken into account. The epiglottis is evolved for purposes of olfaction; the aryepiglottic folds and cartilages of Wrisberg to provide a lateral food channel for the passage of liquids in animals which masticate their food; and the cartilage of Santorini is an important part of the mechanism by which the oesophagus is

suspended and opened. The arytenoid cartilages are described as modified in length for respiratory reasons and the muscles attached to them as concerned in the sphincteric closure of the larynx during deglutition, and in dilatation during respiration; the respiratory movements of the glottis are shown to have a marked effect on the circulation of the blood by altering the intrathoracic pressure. Although all animals have an organ suitable for the production of sound, the extent of voice production depends in the main on the necessities and intelligence of the individual. Some animals, however, and especially those of arboreal habits or arboreal ancestry, are better provided for than are others. In discussing the mechanism of phonation it would seem that variations in pitch of the voice are brought about by means of contraction and relaxation of the thyro-arytenoid muscles; and that the raising of the pitch is due to increased contraction of these muscles, by virtue of which their elasticity is increased, while the rate at which they will recoil after being blown apart is accelerated. This is a different view from that which ascribed to these muscles the function of relaxing the vocal cords, because in the former case the pitch would be raised by contraction of the muscle while in the latter it would be lowered. The attachment of the oesophagus to the cartilage of Santorini has also an important function in the mechanism of phonation.—*The Mechanism of the Larynx, V. E. Negus, Lancet, May 17, 1924, ccvi, 987.*—(*J. S. W.*)

Influence of Position on Diaphragmatic Action.—Hoffbauer and Holzkneckt have shown that when a person lies on the side, the diaphragm on the upper side tends to pull away from the chest-wall and straighten out, losing something of its arch, while the diaphragm on the lower side is crowded cephalad by the abdominal viscera, which tend to slump toward the lower side. On contracton of the diaphragm, as in inspiration, the lower half moves through a much greater distance in the process of straightening out the arch than does the upper half, which remains relatively quiet. In the lateral position the breath-sounds are louder over the base of the lung on the down side than over a corresponding area on the opposite side. The reason for this is the increased diaphragmatic action on the lower side and diminished action on the upper side. This phenomenon may be considered as evidence that the vesicular murmur originates in the parenchyma of the lung, or at least in the finer bronchioles, and not in the larynx, as suggested by Bushnell. The increased sounds on the lower side are often misinterpreted as signifying consolidation, and the diminished or absent breath-sounds on the upper side wrongly supposed to indicate an early pneumonic infection or some pleural involvement. The subcrepitant râles of atelectasis are more frequently heard over the lower than over the upper side. Râles associated with pathological changes are heard best, and sometimes exclusively, on the down side. Functional rest for the lower lobe is best secured posturally by having the patient lie with the affected side up. The peculiar action of the diaphragm in the lateral position is responsible for the facts that (1) breath-sounds are louder over the down side, (2) râles are best heard over the down side, and (3) a lower lobe is most at rest when the patient lies on the opposite side.—*Movements of Diaphragm with Patient in Lateral Posture, T. Howard, J. Am. M. Ass., April 19, 1924, lxxxii, 1242.*—(*B. T. McM.*)

Tuberculosis of Mammalian Tissues in Vitro.—The interrelations between tissue elements and pathogenic microörganisms constitute a problem which can be successfully attacked with the aid of tissue cultural methods. The behavior of cells toward bacteria in a tissue culture can be easily observed in living conditions and followed step by step in its successive stages. With this object in view, the simultaneous cultivation of mammalian tissue and tubercle bacilli was done, using the explantation technique previously described by the author. By this method of tissue culture, one is enabled to duplicate the essential phases of the tuberculous process under artificial conditions. For this the lymphoid tissue of the mesenteric lymph nodes, the omentum, and the loose connective tissue of adult rabbits were chosen. Two strains of human tubercle bacilli were used, one of high and one of low virulence. Clotted blood plasma from young rabbits, with bone-marrow extract, etc., constituted the nutritive media. The nonvirulent bacilli usually completely overgrew the tissue in three weeks. When the virulent strain was used the tissue, on the contrary, finally got rid of the bacilli. By avoiding contamination it was found possible to grow tissue and bacilli together for a long time (3 weeks) without degeneration and death of cells. In the presence of tubercle bacilli the tissue elements always showed a series of definite, specific, reactive changes. In the lymphoid tissue the reticular cells are the most active elements. They are mobilized; they hypertrophy, divide and become transformed into large polyblasts, epithelioid cells,

actively phagocytizing the tubercle bacilli. The epithelioid cells assemble in clusters which could be called "primary" tubercles. Many of the cells fuse together, and in this way giant cells of the Langhans type are produced. The lymphocytes play an active rôle. They hypertrophy, become transformed into polyblasts, and some may acquire an epithelioid character and join the reticular cells in the process of tubercle and giant-cell formation. When lymphocytes are changed into epithelioid cells active phagocytosis of bacilli by them occurs. The tubercle bacilli, engulfed by the epithelioid cells, are either digested, leaving a peculiar yellow pigment, or they multiply excessively in the body of the phagocytes. Such cells, full of bacilli, may keep their vitality for a long time, move about and even divide noticeably. In the giant cells the digestive power of the protoplasm is probably far higher, and therefore the number of intracellular bacilli usually does not increase excessively. Caseation, the typical end result of the tuberculous process, can also be observed in the cultures, for in the larger clusters of "tubercle" the epithelioid cells can be seen to go through degenerative processes similar to the tuberculous process in the body. Similar facts can be observed in inoculated cultures of the omentum and simple connective tissue. In the first, the resting wandering cells are mobilized to epithelioid polyblasts, engulf tubercle bacilli and form typical tubercles. In the second, although the bacilli usually do not penetrate into the tissue, these cells hypertrophy and are transformed into epithelioid and even small giant cells. The fibroblasts do not take part in the formation of tubercles and giant cells, with the possible exception of the omentum. The question of the rôle played by the endothelium in the production of typical tuberculous formations is important. If we confine the use of this name only to the squamous cells lining the common blood vessels—capillaries, arteries, veins and heart—and the lymph vessels, it will be difficult, if not impossible, to find any real proof for their assumed participation in the production of the elements of the tubercle. In the tissue cultures the endothelial cells either keep their former arrangement in tubes and produce new sprouting capillary branches, or they become transformed into strands of common fibroblasts. They never give rise to cells of ameboid, wandering character, to polyblasts. But if we apply the term *endothelium* to all squamous cells in general, lining blood or lymph spaces of any kind, for instance, to the cells of the wall of the venous sinuses of the spleen, to the cells of Kupffer in the liver, to the reticular cells lining the walls of the lymph node sinuses, etc., we shall then have to look upon the epithelioid and giant cells of the tubercles in the organs as products of endothelial transformation. But this would seem a misuse of the term *endothelium*. The elements just mentioned are functionally and structurally quite different from ordinary endothelium in common capillaries, arteries, veins, heart and lymph vessels; on the contrary, ontogenetically and physiologically they are closely connected with the great family of wandering cells. Scattered all over the body, a part of the wandering cells remain in an active and motile condition, whereas others are transformed into fixed, resting, nonmotile elements, but keeping their potentialities of motility in a latent condition. To this last category of cells, potentially motile and ameboid but normally quiescent, belongs a series of elements described by various authors under different names. These include the restive, wandering cells of Maximow, the clasmatocytes of Ranvier, the reticular cells and the cells of the lymph node sinuses, the cells of Kupffer in the liver, etc. Pathologists have largely adopted the name *histiocytes* for the group. Evans gave them the name *macrophages*. By some other writers the whole group is referred to as the *reticulo-endothelial apparatus*. Under the influence of stimulations of various kinds all these cells can be mobilized and then become transformed into motile cells, functioning as active phagocytes, storing vital dyes and various other colloid substances. These resting wandering cells or histiocytes in all their modifications in the different organs and tissues, and not the common endothelial cells, are the active elements in the various inflammatory processes and, in particular, tuberculosis. The tubercle bacilli act on the tissue cells—and chiefly on the resting wandering cells or histiocytes—by means of specific chemical substances, and the action is pronounced in tissue cultures in stages in which no visible disintegration of the bodies of the bacilli themselves can yet be found. The cells may be influenced at a distance without coming in direct contact with the bacilli; the behavior of the cells can therefore by no means by compared with their reaction to inert foreign bodies of the same size and consistence as the tubercle bacillus. The facts observed speak in favor of the theory that a specific chemical stimulation, originating in the tubercle bacilli, causes cell hypertrophy and proliferation directly, without a preliminary injury to the living substance of the cells, causing its degeneration, to be secondarily followed by a reaction of progressive character. Thus, the action

of the tubercle bacilli is a direct specific formative stimulation. Of all the tissue elements, the resting wandering cells or histiocytes, as well as the lymphocytes, are most sensitive. In addition, the presence of the tubercle bacilli seems to have a general stimulating influence on the living substance of various other cells, causing, for instance, the fibroblasts and the true endothelial cells to remain for an abnormally long time in active condition or even to acquire embryonic properties, the fat cells rapidly to lose their fat, the small lymphocytes to hypertrophy, and to easily transform themselves into large lymphocytes, etc.—*Tuberculosis of Mammalian Tissue in Vitro, A. A. Maximow, J. Infect. Dis., June, 1924, xxxiv, 549.—(J. S. W.)*

Infectious and Noninfectious Pulmonary Tuberculosis.—There are active, manifest, cases of pulmonary tuberculosis in which tubercle bacilli cannot be determined during years of observation. Of the cases of pulmonary tuberculosis in which tubercle bacilli cannot be found in the sputum at least 80 per cent do not infect their children. Clinical symptoms alone are not sufficient to determine the infectious nature of a case of pulmonary tuberculosis. The infectious or noninfectious cases are graded as follows: (1) "Open" tuberculosis, tubercle bacilli are found in all or nearly all sputums examined. (2) "Facultative open" tuberculosis: (a) "rarely open" tuberculosis; bacilli are found only rarely or only by animal inoculation, (b) "still closed" tuberculosis; cases with no bacilli in the sputum yet with plenty of râles and marked roentgenological findings, stage III cases, etc. (3) "Closed" tuberculosis; negative for bacilli and without clinical findings, but yet suspected cases.—*Über die Abgrenzung der ansteckungsfähigen Lungentuberkulosen gegen die nichtansteckungsfähigen, Braeuning, Ztschr. f. Hyg., January, 1923, c, 8.—(H. J. C.)*

Enlargement of Pulmonary Lymph Nodes.—The anatomic position of the pulmonary lymph nodes is all important in the consideration of the mechanical effects of their enlargement. The nodes lie in close relationship with the lymphatics, veins arteries and bronchioles, and when enlarged must of necessity press upon these systems of tubes. The results of such pressure will depend, in part, upon the power of resistance of the tubes. The tube with the greatest resisting power will be the bronchiole strengthened as it is by the scales of cartilage. The arteries, and particularly the veins, are the more compressible, and last in order of effective resistance are the lymphatics. Obstruction to the passage of lymph also results from active congestion of the node itself. In pneumonia the enlargement of the nodes at the hilum of the lung is often enormous, and the back-pressure on the veins which drain the parts first affected results in the addition of passive congestion to the active congestion already begun. Perhaps this back-pressure on the veins is an advantage and something analogous to the passive hyperemia afforded by the Bier treatment. The enlargement of the nodes is certainly a factor in the pulmonary edema which sometimes accompanies pneumonia. Delayed resolution of the lung after pneumonia is in large part due to persisting lymph node enlargement obstructing the veins and causing passive congestion. The part played by the nodes in the causation of a pleuritic effusion is a very important one. When there is an inflammatory exudate there is an enlargement of the nodes sufficient to obstruct both lymphatics and veins draining the inflamed parts; there is always found a considerable enlargement of the nodes at the hilum of the lung. Special directions are given for eliciting the area of dulness associated with such enlargement. The author refers to this area as the "oval area of dulness." The relationship between impaired apical resonance in tuberculosis and hilum enlargement is also discussed. Measles and whooping cough are probably the commonest primary causes of chronic enlargement of the intrapulmonary nodes in children. In whooping cough, when fine sticky râles are heard at the bases of the lungs, the oval area of dulness will also be found. Once the nodes have become more or less permanently enlarged, they produce a permanent passive congestion in the area which they govern. The frequency with which bronchiectasis follows whooping cough and measles is in great measure due to this. The influence of the enlargement in favoring hemorrhage in pulmonary tuberculosis must be mentioned. The relief to the patient's breathing after a moderate hemorrhage is more easily explained by the reduction in the size of the nodes than in any other way. In the matter of treatment the application of moist heat (poultices) to the chest is suggested for acute conditions such as pneumonia and bronchitis. In measles and whooping cough a stimulating liniment applied to the bases of the lungs behind is recommended. Iodine and the iodides have also been found useful, especially in the more chronic conditions.—*The Mechanical Effects of Enlargement of the Pulmonary Glands, N. Neild, Brit. M. J., May 17, 1924, no. 3307, 849.—(J. S. W.)*

Duration of Pulmonary Tuberculosis.—Of 370 cases of pulmonary tuberculosis examined in Potsdam 46 per cent died after about $2\frac{3}{4}$ years' duration of the disease, 5 per cent remained incapable of work, and the remaining 49 per cent were able to resume work and, therefore, were clinically cured. Among these the duration of disease was 6 years during the war period and 33 years during peace. A relation between the duration of the pulmonary tuberculosis and age could not be determined. The duration of the disease is associated with the question of the clinical onset of the tuberculosis; if onset is reckoned from the first appearance of phthisis in the sense of Virchow-Orth then pulmonary tuberculosis in man averages a little more than 2 years. It is, however, to be figured that only half of the cases of pulmonary tuberculosis come to the physician's attention late in the course of disease. The other half become clinically healed, and entirely capable of working. Clinical healing can be considered to be of about 8 years' duration. The duration is shortened by the danger of a renewed phthisis. This threatens those having had tuberculosis to a greater extent than it does healthy individuals.—*Zur Frage nach der Dauer der Lungentuberkulose, K. Siegfried, Ztschr. f. Tuberk., July, 1923, xxxviii, 241.*—(*H. J. C.*)

Healed Miliary Tuberculosis of Lungs.—The major roentgen-ray findings of this condition consist of the presence of multiple disseminated discrete shadows of rounded outline and marked degree of density, having sharply bordered edges, all of which are very small in size (not over 3 mm. in diameter). These shadows are spread evenly all over both lung fields, the intervening areas being normally transparent. The degree of shadow density is that found in local calcification of soft tissue structure, such as is often seen in or near the lung root areas in healthy adults. In acute miliary tuberculosis the individual shadows lack this degree of density and definiteness of outline. The pathology of the condition is discussed with references to the literature, and it is pointed out that the miliary tubercles may be disseminated through one or both lungs, or part of a lung. Usually the issue is fatal but occasionally healing occurs. In a majority of instances of the latter type the paucity of clinical symptoms is surprising. Three case reports with reproductions of roentgenograms are included. *Summary:* 1. From postmortem studies reported in the literature it is evident that at rare intervals a miliary tuberculosis of the lungs will heal. Occasionally this healing seems to be accompanied by a calcification necrosis of the individual tubercles, which calcification remains as sole evidence of the disease. 2. The roentgen findings in such cases are small, shot-like shadows, evenly distributed over both pulmonary fields. 3. The distribution of lesions is characteristic of disseminated tuberculosis of miliary type. 4. Although lacking postmortem proof it is highly probable that the 3 cases here reported are to be considered as instances of healed, calcified miliary tuberculosis of the lungs.—*Roentgenological Evidence of Apparently Healed Miliary Tuberculosis of the Lungs, E. S. Blaine, Am. J. Roentgenol. & Radiol. Therap., March, 1924, xi, 233.*—(*J. B. A.*)

Adrenalin Content of Adrenals in Tuberculous Guinea Pigs.—The suprarenal capsules of animals infected with tuberculosis contained less adrenalin than those from normal animals. This was also found to be true in animals given large amounts of bacilli resulting in immediate death.—*On Adrenalin Content in the Suprarenal Capsules in Tuberculous Guinea Pigs, I. Nakada, Kekkaku, March, 1923, i, no. 1, Abstracted from Japan M. World, December, 1923, iii, 274.*—(*M. M.*)

Relationship of Paratuberculosis and Tubercle Bacilli.—It is well known that in the group of acid-resisting microbes there exist in addition to those pathogenic for man and warm blooded-animals (Koch's bacillus, the avian bacillus, Hansen's lepra bacillus, Johne's bovine bacillus of hypertrophic enteritis, and Stephanski's bacillus of rat leprosy), paratubercle bacilli such as the acid-resistant bacilli of cold-blooded animals and acid-resistant saprophytes. These latter are cultivatable both at ordinary and at high temperatures, while true tubercle bacilli cannot stand over 37°C. The acid-fast bacilli of cold-blooded animals (fish bacillus of Dubard, Batallon and Terre, adder bacillus of Sibley, Hansemann's python bacillus, Küster's frog bacillus, the tortoise bacilli of Friedmann and Piorkowski) are pathogenic only for these animals, warm-blooded animals reacting to them only with a caseous focus at the site of inoculation. The saprophytic acid-fast bacilli (smegma, dung, milk, grass and butter) are found in the soil, water, dust, on the skin and mucous membranes, but are entirely nonpathogenic. The question of the relations between all these and true tubercle bacilli has occupied much attention and is related to the problem of vaccination against tuberculosis. Can tubercle bacilli of warm-blooded animals transform themselves into bacilli pathogenic for cold-blooded animals or into saprophytes, or vice versa? This matter has remained

the subject of so much confusion that an attempt is made to clarify the knowledge and facts in our possession. A fairly complete review of the findings of various workers is given and the following conclusions are drawn: Up to the present no experimental fact permits us to affirm that the tubercle bacilli of warm-blooded animals can become transformed into the acid-fast bacilli of cold-blooded animals or into acid-fast saprophytes. The positive results hitherto reported appear, after numerous rigidly controlled experiments, to have been due to superadded infections. However, there is evidence that paratubercle bacilli are related to tubercle bacilli by other bonds than morphological and cultural characteristics. They may act in a measure as antigens in the complement-deviation reaction. This property is also shared with the diphtheria bacillus and B. subtilis. In spite of this antigenic power none of these microörganisms has any immunizing power against tuberculous infection. The paratuberculous group also secrete a tuberculin much less active than human and bovine tuberculin. Although they are not capable of sensitizing the body to true tubercle bacilli, they can give rise to a characteristic reaction in the tuberculous animal. Thus there are undeniable biochemical affinities between the two groups.—*Sur les relations qui existent entre les bacilles paratuberculeux et les bacilles tuberculeux, L. Nègre, Rev. d. l. Tuberc., April, 1924, v, 161.*—(*A. P.*)

Elimination of Tubercle Bacilli in Bile.—Libert and Carnot have previously shown the possibility of obtaining tubercle bacilli in the duodenal liquid withdrawn through a tube. They showed their constant presence in patients with pulmonary tuberculosis with cavity, and that in certain doubtful or suspected cases bilioscopy may furnish proof of their tuberculous nature. At this time it was thought that the bacilli in the bile were not due to ingestion of bacilliferous particles, but to true excretion by way of the biliary and intestinal tract. Nevertheless, there has been some doubt on this point. To support the excretory thesis, three cases of extrapulmonary tuberculous foci were cited, in which the lungs were clinically negative, but radiography was not done. The experimental study of the problem is difficult. A number of further clinical observations are cited. In several cases there were apparently inactive pulmonary lesions, and the sputum even after concentration was negative, yet tubercle bacilli were demonstrable in the duodenal liquid withdrawn by means of an Einhorn tube. One patient had hypertrophic cecal tuberculosis with minimal pulmonary signs and hardly any cough or expectoration. This patient died, and necropsy showed a minimal caseous lesion in the depths of one lung. Close study of cases inclines one to the view expressed paradoxically that "active pulmonary tuberculosis is either open or does not exist." However, although the possibility of the bacilli having been ingested must always remain, biliary elimination is known to accompany bacteremias, temporary or permanent, just as the kidneys may excrete bacilli without renal lesions, as noted by Rist and Kindberg. The work of Freed and Black is reviewed, whose experiments seemed to indicate that there were two independent possible sources of the bacilli; the lung and the liver and biliary tract. Either may exist without the other. —*Nouvelles recherches sur l'élimination du bacille tuberculeux par la bile chez l'homme, E. Libert, Rev. d. l. Tuberc., April, 1924, v, 178.* —(*A. P.*)

Tubercle Bacilli in Blood.—The literature is exhaustively reviewed. The blood of cases of pulmonary tuberculosis in all stages was examined microscopically, and by intraperitoneal inoculation, in 3 cc. amounts into guinea pigs, that were examined every 3 to 4 weeks by intracutaneous injection of 0.1 cc. of 1:5 O. T. according to Römer. After 4 months the guinea pigs were killed and examined microscopically and macroscopically. Of 46 cases examined, the blood preparations were positive 6 times and guinea-pig inoculation 9 times. The tuberculin reaction was negative in 3 cases, in which the pathological finding was definitely positive. The spleen weight of normal guinea pigs is considered 0.1 to 0.19 gm. per 100 gm. of guinea-pig weight; greater weight of the spleen is considered as highly suspicious.—*Tuberkelbacillen im Blut und Römersche Reaktion, F. Ishiwara, Ztschr. f. Tuberk., April, 1923, xxxviii, 86.*—(*H. J. C.*)

Action of Elements of Blood on Tubercle Bacilli.—To extend our immunological knowledge of tuberculosis the proper way is to find out whether the infective agent can be cultivated in the blood, in fluids of the blood, and in the latter in the presence of leucocytes. A general technique, applicable for staphylococcic and streptococcic infections, has been previously described, and this, with certain changes, can also be employed in researches on the tubercle bacillus. The method consists in implanting graduated dilutions of a bacterial culture into the blood, and then filling this implanted blood into capillary tubes, or into slide cells. The latter are shallow cells made from

ordinary microscopic slides held apart and subdivided into compartments by strips of paper of standard thickness. When tubercle bacilli are inoculated in human blood, the hemoglobin of the red corpuscles is unaffected. Simple inspection, therefore, does not, as in the case of staphylococci and streptococci, tell whether a growth has occurred. To determine this, the hemoglobin must be dissolved out before staining and examining under the microscope. This is done by immersing the clots from the slide cells and capillary tubes (capillary clots) in water in which a little acetic acid or saponin is then added. When hemolysis is complete the clots are transferred to slides for fixing and staining for tubercle bacilli. Growth is usually found to be vigorous, being very appreciable after forty-eight hours. A study of the tubercle-implanted clots showed the following: Polynuclear leucocytes are first attracted to the tubercle bacilli and phagocytosis takes place. Destruction of these polynuclear leucocytes results, to be followed by the collecting of the large and small mononuclear leucocytes to form, together with the disintegrating polynuclear phagocytes, large agglomerations around the tuberculous foci. A little later *cavernation* begins, and after forty-eight hours definite cavities are seen, and the process goes on until the slide-cell clots are everywhere perforated with holes. In like manner the tubercle-implanted capillary blood clots are gradually eroded and fall to pieces. The cavernation appears to be due to quite a special chemical action exerted upon leucocytes by the tubercle bacillus. Trypsin, derived from the disintegrated leucocytes, probably acts on the fibrin. Occasionally, where there are many bacilli, cavitation occurs without any apparant intervention of leucocytes. It was found that tubercle bacilli do not proliferate appreciably on serum. However, they grow very freely on plasma (plasma clots) but less vigorously on blood clots. It would seem that the tubercle bacillus obtains from the fibrin of the clot some essential nutrient element which is not present in the serum. Experiments with leucocyte-lined capillary tubes showed in those regions of a plasma clot where a sufficient force of leucocytes (polynuclears) was present, the growth of tubercle bacilli is always very much less copious than where the leucocytes are absent. There is one important exception to this rule: where leucocytes succumb to tuberculous attack, the growth of the tubercle bacillus is distinctly favored. In comparing the bloods from normal people and those with active tuberculosis, it was found that, whereas the leucocytes of normal blood when the blood is heavily implanted collect only sparingly around the tubercle bacillus, the leucocytes of the tuberculous patient congregate in great masses around the bacilli. In the blood of the tuberculous patient there results a much larger dissolution of microbes. The active tuberculous patient has, as compared with the normal, a greatly increased hemo-bactericidal power. This increased power may be conferred on normal blood *in vitro* by the preliminary addition of living bacillary emulsions.— *New Methods for the Study of the Pathology and Treatment of Tuberculous Diseases, A. E. Wright, Lancet, February 2, 1924, ccvi, 218.—(J. S. W.)*

Effect of Guinea-Pig Tissues on Tubercle Bacilli.—Tubercle bacilli in tuberculous lymph nodes kept in physiological salt solution in the incubator for two weeks lost their infecting capacity, but when kept at room temperature or in the icebox they retained their vitality for as long as three months. Bacilli inoculated into normal guinea-pig tissues which were then kept in the incubator under the same conditions, remained infectious after 18 days. If guinea-pig tissues, either normal or tuberculous, be incubated for two weeks and then ground up into emulsions and added to emulsions of virulent tubercle bacilli, they produce death of the bacilli if the resulting mixture is allowed to stand for as long as twenty hours. No effects, however, are produced upon the staining characteristics or the morphology of these bacilli. The effect is not produced by acidity changes. The formation of this toxic product is most rapid when guinea pig's voluntary muscle is incubated (one day) and less rapid with heart, lung, liver, etc. (four days). The substance is thermostable. If it is aerated before being put in contact with bacilli, it is found to retain its toxic effect. It is probably identical with tissue autolysates (Conradi).—*The Effect of Guinea Pig Tissues in Vitro upon Virulent Tubercle Bacilli, H. J. Corper, M. B. Lurie and O. S. Kretschmer, Am. J. Hyg., March, 1924, iv, 77.—(H. S. W.)*

Effect of Chaulmoogra Oil on Cultures of Tubercle Bacilli.—Measured amounts of chaulmoogra oil and its derivatives were added to glycerine-agar media in the process of preparation. The media was later seeded with virulent human tubercle bacilli and the amount of growths on media with varying amounts of oil compared with that on control media and other media containing codliver oil, olive oil or liquid paraffine. Growth of tubercle bacilli is distinctly inhibited by chaulmoogra oil even in quite

high dilutions, while codliver oil produces a very slight inhibiting effect when used in strong concentrations. Other bacteria grow well in chaulmoogra-oil media. Oils related to chaulmoogra also show some specific inhibitory effect upon bacillary growth, as also do the sodium salts of chaulmoogra.—*Chemotherapeutic Experiments with Chaulmoogra and Allied Preparations, O. Schöbl, Philippine J. Sci., December, 1923, xxiii, 533.*—(*H. S. W.*)

Action of Intestinal Juice on Tubercle Bacilli.—Pure intestinal juice from a dog with a duodenal fistula was found incapable of dissolving or digesting tubercle bacilli. After 24 hours the only noticeable effect on tubercle bacilli was a slight decrease in staining power and a definite Much granulization.—*Ueber die Einwirkung reinen Darmsaftes auf Tuberkelbazillen, K. Mylius and F. Sartorius, Ztschr. f. immunitätsforsch. u. exper. Therap., Orig., 1924, xxxix, 12.*—(*E. R. L.*)

New Stain for Acid-Fast Bacteria.—A method, using acetone and iodine-acetone as a decolorizer in the differential staining of tubercle bacilli and of bacterial spores, is described. The technique is as follows: 1. The slide is immersed in steaming carbol-fuchsin (basic fuchsin, 1.5 gm.; 95 per cent alcohol, 15 cc.; carbolic acid crystals, 5 cc.; distilled water, 85 cc.) for 2 minutes or longer. 2. Wash without allowing smear to become dry. 3. Decolorize with acetone or iodine-acetone (1 part of Gram's iodine solution to 3 parts of acetone, or add a few crystals of iodine to acetone) until smear is nearly colorless. This requires only a few seconds. If acetone alone is used and does not decolorize quickly and sufficiently, flood the slide with Gram's iodine solution, immediately drain off, and repeat acetone treatment. Flooding slide with decolorizer twice, tipping back and forth, and washing in water is usually sufficient. Ignore color of thick clumps. Dipping in water at any time will determine whether the slide is properly decolorized. 4. Counterstain with 10 per cent aqueous solution of a saturated alcoholic solution of methylene blue for from one to two minutes, or a saturated aqueous solution of picric acid for from five to ten seconds. The stability of the fuchsin dye depends on the use of high grade chemicals, clean glassware, and pure water. The solubility of the dye in the decolorizer is of considerable importance. Poorly soluble dyes require prolonged exposure to the decolorizer and counterstain. There is no serious objection to staining with boiling carbol-fuchsin provided the smear is not permitted to dry. If the dye becomes dry at a high temperature, it becomes insoluble in the decolorizer, and the smear must be discarded. Acetone decolorizes the smear more rapidly than acid alcohol, and when iodine is utilized, more rapidly than sodium sulphite, and the resulting slide is usually cleaner, owing to a more complete washing of the dye. Acetone or iodine-acetone serve also as a superior decolorizer in the Gram stain. If the iodine-acetone solution is to be used in the Gram stain, crystals instead of Gram's solution should be added to the acetone as a small amount of water in the acetone affects the Gram reaction. The method described for the staining of tubercle bacilli, slightly modified, serves as a superior spore stain. The slide is not allowed to dry before cooling and decolorization is done with acetone. Loeffler's methylene blue is preferred as a counterstain. By this method, living spores show only marginal staining, whereas spores killed by heat stain to the centre.—*A New Method of Staining Acid-Fast Bacteria and Spores, V. Burke and M. Dunning, J. Infect. Dis., February, 1924, xxxiv, 105.*—(*J. S. W.*)

Constituents of Tubercle Bacilli Extractable by Ether.—*On the Constituents of Tubercle Bacilli Extractable by Ether, Y. Takeda, Osaka Igakkwai Zasshi, October, 1923, xxii, no. 10, Title from Japan M. World, March, 1924, iv, 74.*—(*M. M.*)

Homogeneous Cultures of Tubercle Bacilli.—By cultivating tubercle bacilli in a medium consisting of 4 per cent glycerol-agar and containing alkaline yolk of egg, a wet film of the bacilli proliferates, and by repeated cultivation in this manner a homogeneous cultivation may be obtained. It is believed to be due to the egg-yolk albumin. Some strains are more easily made to grow homogeneously than others; two human, one bovine and one avian stain were thus grown. By employing the agglutination test with these cultures a clear distinction was obtained between the human, bovine and avian bacilli.—*On the Homogeneous Cultivation of Tubercle Bacilli and a Study with the Tubercle Bacillus Obtained through Homogeneous Cultivation, R. Kawamura, Keio-Igaku, May, 1923, iii, no. 5, Abstracted from Japan M. World, December, 1923, iii, 281.*—(*M. M.*)

Glucose Formation by Tubercle Bacilli.—Tubercle bacilli cultivated on albumin-free media produce reducing substances. They were proved not to be decomposition products of the bacilli or the bacillary bodies itself, but the metabolic products of the bacilli. Glycerine can be

changed into glycerose, or dioxyacetone or glycerine aldehyde, and a combination of these two decomposition products might produce glucose. It is interesting to note that tubercle bacilli can produce glucose by decomposing glycerine and then the sugar can be utilized for nourishment. This reducible substance might also be produced under unfavorable environments such as exposure to diffuse sunlight, or when film of culture is precipitated in liquid media.—*Biological Studies of Tubercle Bacillus. I. Communication. On Glucose Formation of Tubercle Bacillus, J. Ohnawa, Osaka Igakkwai Zasshi, November, 1924, xxii, no. 11, Abstracted from Japan M. World, March, 1924, iv, 74.*—(*M. M.*)

Tuberculosis Immunity, Tuberculin Sensitiveness and Allergy.—Immunity in tuberculosis has been termed "relative immunity" and "immunity of infection." Selter's investigations indicate that the degree of immunity is not proportional to the extent of infection. Animals with single tuberculous nodules are more immune than animals with extensive disease. In the human being the situation is doubtless similar; the tuberculization of the race, which in recent times has led to higher immunity, has been for the most part a matter of slight infection of lymph nodes and viscera. The immunity differs from the immunity of other diseases, even syphilis, to which the resemblance is closest. Tuberculosis immunity occurs only in the event of actual infection with living tubercle bacilli and persists only as long as living bacilli are present. With complete healing immunity vanishes. The same is true of syphilis. However, in syphilis such clinical healing with complete loss of immunity is a common event, whereas tuberculous nodules do not tend to complete healing, but persist as such to the end of life. The situation in tuberculosis is further complicated by the fact that two kinds of infection occur, human and bovine, that is, native and foreign types. The human being is susceptible to infection with the foreign, bovine type, but such infection tends to die out, just as human-type infection of animals dies out in the course of a few months or years. Nothing exact is known of the mechanism of tuberculosis immunity, further than that it is intimately bound up with the cells of the body and is not a matter of humoral antibodies. Tuberculin sensitiveness stands in the closest relation with tuberculosis immunity. The former, like immunity, is produced only by actual infection, not by the introduction of killed bacilli. Exact observation, however, shows that the development of immunity precedes the tuberculin sensitiveness. Without immunity there is no sensitiveness. There is, however, no parallelism between sensitiveness and immunity. In fact Selter's animal experiments show that animals with slight tuberculous changes and scarcely demonstrable tuberculin sensitiveness have the highest degree of immunity. The tuberculin reaction is in no sense an anaphylactic response. Tuberculin is a specific irritating substance which calls forth a reaction only in the infected host, needs no antibody to permit or promote its action, and, as far as present evidence goes, need not itself be changed in the course of the reaction. The term "sensitiveness" to tuberculin is correct, and "hypersensitiveness" incorrect. True hypersensitiveness to the protein of the tubercle bacillus can be produced, but this is not the condition ordinarily meant by tuberculin sensitiveness. The term "allergy" leads to numerous complexities unless qualified, as it is by many authors, according as tuberculin sensitiveness or tuberculosis immunity is meant. It is best to refrain from the use of the term "anergy" altogether, in view of the lack of parallelism between tuberculin sensitiveness and immunity. The cells of the noninfected animal are insensitive to tuberculin, but susceptible to tubercle bacilli, while the reverse is true of the infected. Tuberculin sensitiveness is increased in progressive tuberculosis and decreased with healing. This decrease is associated with an increased immunity. On the other hand, a decrease is also observed in pregnancy, menstruation and other conditions, which appears to be associated with decreased immunity. Selter recommends the terms "positive" and "negative reduced tuberculin sensitiveness," to indicate decline in tuberculin sensitiveness, respectively, with increased immunity and decreased immunity. The term "allergy" should be reserved for the manifestations of immunity in chronic infection.—*Tuberkuloseimmunität, Tuberkulinempfindlichkeit, tuberkulöse Allergie, H. Selter, München. med. Wchnschr., April 11, 1924, lxxi, 462.*—(*E. R. L.*)

Allergy to Tuberculins Derived from Saprophytic Acid-Fact Bacilli.—Turtle, water and butter bacilli were used for preparing tuberculin with which, besides O. T., 688 intracutaneous injections were given 50 patients. O. T. and a saprophytic bacillus tuberculin were injected in amounts of 0.1 cc. of 1 to 10 dilution in the same arm. Readings made from the first to fifth days were tabulated. A definite quantitative or qualitative difference in the action of the saprophytic tuberculins was not determinable. There was, however, a definite paral-

lelism between the reaction to O. T. and the saprophytic tuberculins, in that an allergic tuberculous body reacted strongly to all and an anergic one weakly to all. Therefore, there is an elective irritative action of the tuberculin from acid-fast saprophytes on the tuberculous body. This relationship was lacking between nonspecific bodies (diphtheria, coli, toxin and horse serum) and O. T., indicating that there are basic differences between O. T. and nonspecific irritants. —*Die Reaktion des tuberkulösen Organismus auf intracutane Verimpfung von Alttuberkulin, von Tuberkulin säurefester Saprophyten und von unspezifischen Körpern; E. Lange, Ztschr. f. Tuberk., July, 1923, xxxviii, 334.*—(*H. J. C.*)

Allergy in Tuberculosis and Syphilis.—Allergy may be defined as the hypersensitiveness that a body infected by a microbe shows to this microbe and its toxins. Koch's phenomenon is a good illustration. In human tuberculosis the phenomenon of specific allergy may be shown by the introduction of tuberculin into the tissues. The reactions appear shortly after infection and disappear when resistance flags, as during acute exacerbations or tuberculous cachexia. Certain intercurrent affections weaken the reaction, such as measles and secondary syphilis. These same maladies are known to aggravate tuberculosis. Noguchi, after cultivating the treponema of syphilis, hoped to be able to establish a skin test similar to the tuberculin test, but further investigations have yielded discordant results. Syphilitic allergy does not develop in the same way as that to tuberculosis, although the tertiary lesions are comparable to Koch's phenomenon. Syphilitic allergy evidently appears much later than is the case in tuberculosis, tertiary lesions rarely occurring before the third year, often much later and sometimes never. The luetin reaction has been shown by Desneux to occur characteristically only in tertiary syphilis. Here allergy appears with extreme violence and the skin reactions are apt to be characterized by necrosis at the site of inoculation. To sum up, there exists in tuberculosis an almost constant specific allergy, elicited by a specific product, tuberculin; there exists in syphilis a specific delayed and irregular allergy, as transitory as the tertiary manifestations themselves, manifesting itself among other ways by the luetin skin reaction; luetin, however, is not a pure product, nor is its specificity comparable to that of tuberculin. There are also a number of interesting associated phenomena. There is a certain parallelism between the intracutaneous tuberculin reaction and the reaction to certain other substances such as serum, cow's milk, etc. Thus, by the side of specific allergy or hypersensitiveness there exists a nonspecific or hetero-allergy. A similar allergy can explain the positive skin reactions obtained in syphilis by such substances as the culture media without the spirochetes, and the positive fixation reactions with extract of normal liver (Neisser and Bruck). Thus, there may be an allergic terrain capable of reacting to other than specific irritants, in other words, a polyvalent allergy. Such a terrain will react violently to certain infections and intoxications. It is interesting to study syphilitic allergy from a similar viewpoint. The allergic syphilitic is apt to react violently to tuberculin, given intracutaneously. Clinically, the reciprocal influence of these two affections is well recognized, and antisyphilitic treatment may affect favorably tuberculous manifestations in a syphilitic person. Bazin's erythema nodosum, occurring in a syphilitic, may likewise clear up following neosalvarsan. Thus our original conceptions of allergy must be modified as follows: The introduction into the body of an antigen evokes a specific allergic response, and likewise a nonspecific hetero-allergy, which reinforces the other and reacts upon it. The secondary manifestations of syphilis have a peculiarly aggravating influence on pulmonary tuberculosis. During this phase the body is anergic. During the allegric phase the reverse may occur. In certain old cases, when allergy wanes clinical benefit may be obtained by inducing reactions, as by means of tuberculin. Again, potassium iodide attenuates skin hypersensitiveness and effaces the tertiary lesions of syphilis; but this same allergy is a potent weapon of defense; hence the encouraging results of protein therapy which, however, have not been as satisfactory in tuberculosis. The best method of utilizing nonspecfic allergy is by subcutaneous injections of milk, which may be tried in general paralysis, supplemented by tuberculin injections.—*L'allergie dans la tuberculose et la syphilis, B. Dujardin and C. Duprez, Rev. Belge d. l. Tuberc., March, 1924, no. 3, 87.*—(*A. P.*)

Experimental Cutaneous Hypersensitiveness without Tubercle.—Since the observation by Koch of the so-called "Koch phenomenon," the allergic reaction of tuberculous animals to the tubercle bacillus or its products has been a matter of the greatest interest to students of tuberculosis. Experience has shown that while immune serum reactions and a general anaphylactic reaction to tuberculin can be elicited in animals immunized or sensitized with some form of

tubercle-bacillus protein, the cutaneous hypersensitiveness to tuberculin is associated with an infection with live tubercle bacilli. It is believed that a definite focus is always present somewhere in the body if a definite skin reaction can be elicited, and that the latter appears with the development of tubercle and fades with its absorption. If the focus as such is a factor in the production of skin hypersensitiveness to tuberculin it might conceivably be possible to obtain preparations from it which would influence the hypersensitization. The following observations are reported from experiments carried out with the view of throwing light on such a possibility. Tuberculous guinea pigs treated intraperitoneally with filtered extracts of tuberculous foci gave a more virulent reaction than the control when tested intracutaneously with water extract of tubercle bacilli. Normal guinea pigs treated intraperitoneally with filtered extracts of tuberculous foci or of sterile inflammatory foci develop skin hypersensitiveness of the tuberculin reaction type to water extract of tubercle bacilli. The cutaneous response to tuberculin of infected animals and of those sensitized with filtrates of focal tissue extract may be indistinguishable. The latter tend, however, to give a greater proportion of large, bright red, boggy reactions, and have never shown hemorrhage or necrosis. Whether this difference is inherent or eliminable by proper dosage of filtrate is not determined. Animals bearing sterile inflammatory foci show no cutaneous tuberculin reaction. Tissue destruction as shown in histological sections from a series of tuberculous guinea pigs follows rather than antedates the appearance of cutaneous hypersensitiveness. Focal tissue formation may antedate hypersensitization.—*Cutaneous Hypersensitiveness to Tuberculin in Guinea Pigs, L. B. Lange, J. Med. Rev., March, 1924, xliv, 293.*—(*J. S. W.*)

Tuberculin Hypersensitiveness without Infection.—Until a very short time ago it was generally accepted that typical tuberculin hypersensitiveness in guinea pigs, as elicited by skin reactions, could not be produced without the presence of living tubercle bacilli in the body of the animal. Recently, however, the authors have succeeded in obtaining definite, though transitory, skin hypersusceptibility in animals energetically treated with considerable amounts of the nucleoprotein extracted from ground tubercle bacilli. Elaboration of these experiments now indicates that appropriate treatment with tubercle bacilli killed by heat will produce skin sensitiveness to tuberculin indistinguishable in all essential respects from that observed in tuberculous guinea pigs. The following conclusions are presented: *1:* Tuberculin skin reactiveness, both to residue antigens and to old tuberculin, can be obtained in guinea pigs by injections of dead tubercle bacilli killed by heat. *2:* Heating to 100°C. does not destroy the properties of the bacilli which induce this reaction. *3:* If sufficient amounts are injected the reactions appear almost as rapidly as they do after an injection of living bacilli and quite as intensively. 4. So far as can be judged at the present time there seems to be a general parallelism between the extent of the local reaction to the injected dead bacilli and the degree of tuberculin sensitiveness obtained.—*Tuberculin Hypersensitiveness without Infection in Guinea Pigs, H. Zinsser and S. A. Petroff, J. Immunol., March, 1924, ix, 85.*—(*J. S. W.*)

Cellular Immunity and Predisposition to Disease.—Variations in cellular immunity are weighty factors in the development and treatment of many diseases. In tuberculosis, besides disposition and increased resistance due to earlier infections, the behavior of the connective tissues, their mineral (especially silicic acid) content and the increased cellular content resulting from increasing age play a part. The lung of older individuals possesses, besides the anthracotic areas resulting from the wandering in of small foreign bodies, an increased and extended perivascular and peribronchial stroma, with rich lymphocytic deposits which tend to oppose the development of the tubercle bacilli. The lack of these circumstances in the organs of younger persons and certain individual groups, explains the more easy and more severe infection of their lungs. Also, the infections overcome in youth call forth inflammatory reactions and, with these, increased cellular immunity. The same is true of the vascular changes. While sclerosis is a purely physiological change occurring with increase in age, atheromatosis is a pathological process with necrosis in the vessel walls and all its consequences. Carcinoma is discussed in the same light. Cellular reinforcement is obtained by an artificially produced inflammatory reaction (diathermy, venesection, injection of lymphocytic substances, skin and gastrointestinal irritation, and roentgen raying). These methods have yielded good results in the treatment of carcinoma especially.—*Celluläre Immunität und Krankheitsdisposition, A. Theilhaber and H. Rieger, Deutsche. Ztschr. f. Chir., 1922, clxxiii, 78.*—(*H. J. C.*)

Lack of Specificity in Response of Host.—An attempt was made to determine

whether tissue response in tuberculosis was more marked to one's own strain of tubercle bacilli than to strains from other patients. The sputum of 4 patients was treated with antiformin and centrifugated. The sediment was thoroughly washed and heated to 70° for an hour in carbol-NaCl. Each patient was tested with the sediment from all four sputums, such precaution as was possible being taken to ensure equalization of dose. No evidence was secured indicating that any patient responded more vigorously to his own strain than to the others. A patient responding feebly to his own strain made weak response to all others, and patients reacting actively to their own strains made fully as intense response to the others.—*Zur Frage der Wirtsanpassung, Grete Freyman, Ztschr. f. Immunitätsforsch. u. exper. Therap., Orig., 1924, xxxix, 1.—(E. R. L.)*

Different Constituents of Tuberculin in Experimental Tuberculosis.—Three preparations were made from old tuberculin: *A:* the precipitate from O. T. with ammonium sulphate treated with pancreatin; *B:* the precipitate from O. T. by means of alcohol treated with pancreatin; and *C:* the portion of O. T. not precipitated by alcohol digested with pancreatin. Comparative tests were made using these three preparations and O. T. itself and it was found that *C* gave the most satisfactory results. *C* was also used with success in treating skin tuberculosis.—*Influences of each Different Constituent of Tuberculin in Experimental Tuberculosis. A Supplementary Report. On the Influence of the Constituents of Tuberculin against Skin Tuberculosis, K. Yoshizawa, Jikkenigaku Zasshi, April, 1923, vii, no. 4, Abstracted from Japan M. World, February, 1924, iv, 47.—(M. M.)*

Hypersensitiveness of Skin to O. T. and Bovine Tuberculin.—Intracutaneous tests demonstrate the superiority of O. T. over bovine tuberculin in diagnosis. The Pirquet test is not suitable for comparative tests. The figures obtained agree well with the prevalence of the bovine type of tuberculosis in man as determined by animal inoculation. In skin tuberculosis 16 per cent was obtained, double that seen among other cases (8 per cent).—*Vergleichende Untersuchungen zur Frage der Empfindlichkeit der Haut gegenüber Alt- und Perlsuchttuberkulin mit besonderer Berücksichtigung der Hauttuberkulosen, H. J. Markert, Beitr. z. Klin. d. Tuberk., April, 1923, lv, 94.—(H. J. C.)*

Mode of Action of Tuberculin.—Selter and Tancré attribute the local tuberculin reaction, the tissue reaction, to tuberculin, while Hayek and Wieser attribute it to the intermediate split products. Such facts that after larger doses there is hardly perceptible reaction but that on the other arm a painful infiltration occurs, or there is full appearance of a tissue reaction only after 24 hours, cannot be explained by these theories. The following working hypothesis is put forward: Tuberculosis immunity depends upon the cellular reactivity of the body cells. The result of this reactivity is that tuberculin causes the formation of immune substances. The antibody-forming function of the cells is expressed in the inflammatory reaction of the tissues. The result of this primary cell activity is the appearance of split products, which, if not sufficiently rapidly and completely decomposed, as intermediate substances have the power to cause general symptoms. The cellular tissue symptoms are, as such, produced by tuberculin, while the fever is elicited by the intermediate split products.—*Die Wirkungsweise des Tuberkulins, E. Szász, Ztschr. f. Tuberk., July, 1923, xxxviii, 342.—(H. J. C.)*

Immunization in Tuberculosis.—*Studies on Immunization in Tuberkulosis. VII Communication. On the Therapeutic Results with Our Preparation "A. O.," R. Arima, K. Aoyama and J. Ohtsuna, Kekkaku, July and October, 1923, i, nos. 4 and 5, Title from Japan M. World, February, 1924, iv, 45.—(M. M.)*

Immunization in Tuberculosis.—*Experimental Studies on the Infection and Immunization of Tuberculosis, J. Hiroshige, Kekkaku, June, 1923, i, no. 3, Title from Japan M. World, February, 1924, iv, 45,—(M. M.)*

Immunization in Tuberculosis.—*Experimental Studies on the Infection and Immunization of Tuberculosis (concluded), T. Hiroshige, Kekkaku, July, 1923, i, no. 4, Title from Japan M. World, February, 1924, iv, 45.—(M. M.)*

Immunization in Tuberculosis.—*Experimental Studies on the Infection and Immunization of Tuberculosis, I. Hiroshige, Kekkaku, October, 1923, i, no. 5, Title from Japan M. World, February, 1924, iv, 45.—(M. M.)*

Immunization in Tuberculosis.—Tubercle bacilli seem to become more and more virulent with persistence in the body of the experimental animal (guinea pig). In 9 days the injected bacilli had a maximal virulence.—*Experimental Studies on the In-*

fection and Immunization of Tuberculosis (concluded), J. Hiroshige, Kakkaku, February, 1924, ii, no. 1, Abstracted from Japan M. World, May, 1924, iv, 127.—(M. M.)

Immunization in Tuberculosis.—The cultivation of tubercle bacilli on saponin medium resulted in obtaining an attenuated strain, the virulence being reduced to 1/1000 of its original. From this culture a vaccine was prepared.—*Studies on Immunization against Tuberculosis. III. Specific Prophylactic and Therapeutic Agent of Tuberculosis. R. Arima, K. Aoyama and J. Ohnawa, IV. Test of Virulence of Our Tuberculosis Vaccine against the Animal, J. Ohnawa, K. Aoyama and R. Arima, Kekkaku, March, 1923, i, no. 1, Abstracted from Japan M. World, December, 1923, iii, 274.—(M. M.)*

Effect of Cholera Vaccine on Tuberculous Cases.—*On the Influence of Cholera Vaccine against Tuberculous Cases, T. Mizobuchi, Kekkaku, March, 1923, i, no. 1, Title from Japan M. World, December, 15, 1923, iii, 274.—(M. M.)*

Immunization of Rabbits against Tuberculosis.—By treating human tubercle bacilli with saponin and lipase they were made nonacid-fast and a highly attentuated strain of bacilli was obtained, which was used for making a vaccine. This was administered intravenously in experimental animals. Infection experiments with animals thus vaccinated revealed in the vaccinated animals less severe foci, while those given smaller infecting doses had practically no foci. It is concluded that the nonacid-fast vaccine obtained by means of saponin and lipase has conferred an immunity against tuberculous infection.—*Studies on Immunization for Tuberculosis. V. Experimental Inoculation with Tuberculosis Vaccine. Part I. Inoculation into the Rabbit, T. Onawa, Kekkaku, May, 1923, i, no. 2. Abstracted from Japan M. World, January, 1924, iv, 19.—(M. M.)*

Erythrosin Tuberculin.—*On Immunization for Tuberculosis and Erythrosin Tuberculin, Y. Watanabe, Kekkaku, May, 1923, i, no. 2, Title from Japan M. World, January, 1924, iv, 19.—(M. M.)*

Antigenic Action of Lipoids of Tubercle Bacillus.—Tubercle bacillus lipoid has a specific antigenic power, unlike other lipoids. —*On the Antigenic Action of Tubercle Bacillary Lipoid, Y. Fujisawa, Kekkaku, October, 1923, i, no. 5, Abstracted from Japan M. World, February, 1924, iv, 46.—(M. M.)*

Hepatic Changes Due to Combined Action of Tuberculin Toxin and Alcohol.—Alcohol and tuberculin toxin were injected in different combinations and by different methods. If the tuberculin toxin was injected first, and then alcohol repeatedly, there developed irregular patches of cirrhosis in the liver. Proliferation of connective tissue as seen in Laennec's cirrhosis was, however, never observed.—*On the Changes of the Liver Due to the Combined Actions of Tubercle Toxin and Alcohol, G. Kachi and B. Midorikawa, Aichi Igakkwai Zasshi, November, 1923, xxx, no. 6, Abstracted from Japan M. World, February, 1924, iv, 37.—(M. M.)*

Symptoms of Pulmonary Tuberculosis.—This study deals with 59 incipient and 278 moderately advanced cases admitted to the Essex County (Massachusetts) Sanatorium. Cough is the most common symptom. Productive cough occurred in a little more than half of the early cases. Tubercle bacilli are not present in the sputum unless ulceration of a focus of disease occurs communicating with the air passages, and continues as long as the ulceration remains open. In the other cases physical signs, X-ray, or a focal tuberculin reaction must prove the presence of pulmonary tuberculosis. Constitutional symptoms show the presence of clinical tuberculosis, and their severity shows the degree of toxemia. The weight under ordinary conditions of life, susceptibility to fatigue, the presence of afternoon fever and rapid pulse, are the foundations for making a diagnosis. The time has passed when it is necessary to defend rest, both local and general, in the treatment of active tuberculosis, whether the rest be a retention apparatus for joint, pneumothorax for lung, or whispered voice for the larynx. —*Symptoms of Pulmonary Tuberculosis: Their Origin and Frequency, O. S. Pettingill, Boston M. & S. J., February 14, 1924, cxc, 139.—(B. T. McM.)*

Dyspnea in Pulmonary Disease.—Dyspnea is the conscious desire for increased pulmonary ventilation. But increased pulmonary ventilation may occur without conscious desire. The animal body possesses marked powers of adaptation. This is amply illustrated by the acquired capacity of aviators and mountaineers to attain altitudes, and even accomplish severe physical exertion, in such a rarefied atmosphere as some authorities had predicted was theoretically impossible without certain artificial aids. The effects of acute oxygen want is to produce a moderate degree of dyspnea which is not so much due to lack of oxygen as to the fact that the body has not sufficient time

to lower the hydrogen-ion concentration of the blood by removing carbon dioxide. If there is a more insidious oxygen want there is no demand for violent pulmonary exertion. The respirations, however, will become gradually shallower, until eventually the inspiration and expiration are so diminished that the respiratory movement practically ceases. This is the anoxemic type of respiratory failure. The asphhxial type is when, because of resistance to inspiration and expiration, the respiratory effort becomes more violent until a sudden climax is reached, when it becomes excessively rapid, and shallower until it ceases altogether. Respiration, besides being controlled by the relative proportions of carbon dioxide and oxygen in the tissues, is also influenced in a profound degree by a nervous reflex. In lesions producing obstruction in the larynx or trachea the asphyxial type of respiratory failure occurs. In conditions causing diffuse bronchial obstruction, such as spasmodic asthma, a more or less pronounced anoxemia occurs. In lobar pneumonia there is an anoxemia due to pollution of the general circulation by nonaerated blood coming from the consolidating lungs. The urgent dyspnea and cyanosis in bronchopneumonia are due to the presence of a carbon-dioxide retention as well as an oxygen retention. The dyspnea in chronic alveolar lesions, such as emphysema, is not very marked, as, due to the insidious development, the body adapts itself. In fibroid phthisis the occurrence of dyspnea depends on the condition of the lungs. If there is complete fibrosis of a part, with obliteration of the circulation and no surrounding inflammatory area, there may be few signs of dyspnea. In large areas of fibrosis or partial atelectasis the circulation may continue to some extent. This blood will not be properly aerated, and on joining the general arterial blood it will reduce the oxygen saturation and increase the carbon-dioxide control of the latter. The severity of the dyspnea and other symptoms which result will depend upon the proportion in which the aerated blood be diluted with the nonaerated blood. Similar conditions occur in extensive pulmonary tuberculosis.—*The Cause and Significance of Dyspnea in Pulmonary Disease, J. Meakins, Brit. M. J., April 5, 1924, no. 3301, 613.—(B. T. McM.)*

Curious Case of Pulmonary Hemorrhage.—This man, admitted to the English Sanatorium, Montana, Switzerland, aged 41, had had frequent and repeated hemoptyses, at intervals of ten to fourteen days. Tubercle bacilli had never been found in the blood spat up, and physical signs in the chest were practically normal with only a few fine distant râles at the right base and occasionally at the left base. The X-ray picture showed a heavy right hilum shadow. He never had fever or evidence of toxemia. He had been gassed with mustard gas in March, 1918. Despite all care and treatment the hemoptyses became larger and it was finally decided to induce pneumothorax on the right side. This apparently checked further bleeding. The three possible diagnoses which suggest themselves are (1) tuberculosis, (2) some nontuberculous lesion, possibly resulting from gassing, (3) syphilis of the lung. The second diagnosis is considered most probable.—*A Curious Case of Pulmonary Hemorrhage, J. Hudson, Tubercle, February, 1924, v, 214.—(A.P.)*

Blood Pressure of Tuberculosis Patients.—*Contribution to the Knowledge of Blood Pressure of Tuberculous Patient. I. Relation Between Blood Pressure and the Site of Infection. II. On a New Method of Estimating Blood Pressure, and the Practical Estimation of Blood Pressure with This New Method on the Patient Having Night Sweats and Contribution to the Pathology of Night Sweats, R. Takahashi, Kekkaku, May, 1923, i, no. 2, Title from Japan Med. World, January, 1924, iv, 19.—(M. M.)*

False Febrile Reaction to Tuberculin.—It has long been known that certain people with a neurotic or hysterical tendency will give a febrile reaction to acupuncture or to the injection of sterile physiological salt solution. Occasionally, such a reaction occurs after the injection of tuberculin and temperature elevation in itself after tuberculin injection is not a sufficient criterion upon which to determine the specificity of the response.—*Ueber nicht spezifische Reaktionen bei der spezifischen Diagnose und Therapie der Tuberkulose, L. Petschacher, Wien. klin. Wchnschr., November 15, 1923, xxxvi, 812.—(H. S. W.)*

Axillary Temperature in Tuberculous Cases.—*On Arm Pit Temperature of Tuberculous Cases, S. Endo and K. Murao, Kekkaku, March, 1923, i, no. 1, Title from Japan Med. World, December 15, 1923, iii, 274.—(M. M.)*

History of Percussion and Auscultation.—This is an address on the work of Auenbrugger and of Laennec. Auenbrugger's fame rests on the few paragraphs in his book, "Inventum Novum," in which he announces the discovery of percussion, states where in health the note is sonorous and where it is dull, and teaches us in what

diseases it is dull. Laennec may be said to have been one of the founders of the clinical-morbid anatomy method of studying disease which, although other methods have since been added, still remains the cornerstone of medicine.—*The History of Percussion and Auscultation, W. Hale-White, Lancet, February 2, 1924, ccvi, 263.*—(*J. B. A.*)

Human Thorax as Resonator.—All resonators respond to various tones, but particularly to those nearest their own fundamental tone. There is thus said to be a "zone of resonance." Vibrations induced in a resonator which differ from its fundamental tone are said to be forced on it. Such vibrations are of much importance in musical instruments, which are so constructed as to reinforce given tones. The sounding board of a piano is a resonance chamber. The violin is a still more conspicuous example. The tones of the strings are forced on the violin body, made of very sensitive vibratile wood. Tones may be forced on a resonator either by supplanting its fundamental tone, as in the case of the reed organ, or by supplementing its fundamental tone, as when a series of wooden boxes of varying size and shape are struck by a hammer, or when two pieces of metal held against the boxes are struck. This last experiment is more or less analogous to forcing tones of phonation on the human thorax. In this we have a complex of many tones of varying pitch, the predominant tones varying with the boxes. The thorax reasonator of course, is not hollow, but multilocular, and its solid contents are under a certain definite tension, that is, it is a "compound resonator," or a large resonator containing a smaller one surrounded by insulating material. The insulation varies in thickness, and the transmitted sounds vary likewise. The percussion note is noticeably higher over the upper thorax, where the larger bronchi are thinly covered. The normal percussion note is a composite of high tones from the impact of the blow on the plexímeter and deep tones of thoracic resonance. Martini found the percussion note of the exenterated and inflated lung of the same pitch as before removal from the chest, showing that high-pitched tones are not forced on the lung. The lung, however, is a very sensitive resonator for sounds within its range. The chest-wall vibrates to the sound waves returned from the lung, as well as those which pass through its own tissues without penetrating. They cannot, however, institute independent vibrations because these are damped by the lung. Bushnell contradicts the opinion that vibrations of the bronchial system excite the vesicular murmur, but believes that other tones, masked over the bronchi by their own fundamental tone, are transmitted to and reinforced by the lung. He regards the air tubes as speaking tubes, down which are carried various laryngeal sounds which excite free vibrations wherever they go. Von Müller has recognized the tones of the vesicular murmur in tracheal breath sounds. Bushnell, however, agrees with Martini in excluding the smallest bronchi and bronchioles from the rôle of resonators, as Sahli thought, because their diameter is too small to permit resonation. Martini's objection that relaxed vocal cords could not play a rôle in the production of bronchial breathing is answered by the statement that they are not completely relaxed in ordinary breathing, but flutter in the air current, and therefore produce sound as would a wet handkerchief fluttering in a breeze. Martini likens the mechanism of tracheal breathing to that of a lip-pipe which is substantially a resonator. The chief difference between his and Bushnell's viewpoint is that Martini thinks the edges of the bronchial bifurcations represent the lip: Bushnell grants that this may be the case in "reciprocating breathing" with the glottis closed and the air under positive pressure, and disclaims the view that resonation cannot be aroused by excitants other than vibrations of the vocal cords; but he believes that under normal conditions the vocal cords are the principal resonators, and that his experiments elsewhere described on breathing with the glottis relaxed prove this. To this argument Martini opposes the negative evidence that he always hears breath-sounds over both trachea and lungs, when respiration is rapid enough. Bushnell replies that increase in rapidity of breathing makes it difficult to keep the larynx open, and may possibly cause sound in a new way, as Beau observed when respirations were increased beyond 45 per minute. Bushnell has observed that when the larynx is silent, the lung is also silent, and when it produces sound, the lung does also, although not necessarily the vesicular murmur. If Martini hears bronchial breathing over the trachea and the vesicular murmur over the chest when the tracheoscope excludes the vocal cords, the explanation is that the high-pitched notes of the tube-sounds are resonated in the trachea and larger bronchi, and probably also in the pharynx, while over the chest only the lower tones are resonated. The production of sound by blowing through a tube is well known. Another source of sound is the escape of air from a small tube or space to one of larger dimensions. This is the cause of those adventitious sounds heard when the subject does not breathe

properly. The demonstration of the specific vibratility of lung tissue is one of the most important contributions to our knowledge of acoustics of the lung. The construction of the thorax practically divides it into two separate chambers or resonators, so that vibrations of the one side are not readily transmitted to the other. Also, owing to the multilocular construction of the lung, the percussion stroke tends to be localized in its effects, yet owing to the specific vibration of lung tissue its tone is the fundamental tone of the thorax. The pitch of the lung note also varies with the size of the lung; hence it is deeper in men than women, higher yet in children. The bronchi and connective tissue envelope of the air-cells tend to check and reflect vibrations similarly to the chest wall. The increased weighing of lung tissue takes place *pari passu* with their growth; we may compare them to piano strings wrapped with wire, which deepens their tones. High-pitched vocal sounds are resonated only in the bronchi, while the deeper bass notes include the fundamental tones of the thorax. Geigel's and Sahli's theories of origin of the vesicular murmur are untenable. Martini's more recent explanation is somewhat vague, but based on his theory of specific vibrations of lung tissue, the vesicular murmur being compared to the sound produced by an alternately tautened and relaxed violin string. Bushnell believes that he has demonstrated, however, that the imposition or reinforcement of vibrations transmitted from the upper respiratory tract is an explanation more in accord with the observed facts. In disease, generally speaking, it is then the transmission, not the character of breath-sounds, which is altered. —*The Human Thorax Considered as a Resonator, G. F. Bushnell, Arch. Int. Med., June 15, 1924, xxxiii, 763.*—(*A. P.*)

Harsh Breathing.—Two forms of harsh breathing are differentiated. The pulmonary harsh breathing is differentiated into impure and muscular harsh breathing. The former is explained as occurring at the end of inspiration in the smaller pulmonary divisions during successive rapid respirations, and resulting from inflammatory conditions with tenacious secretions or swelling of the mucous membranes. The second form is associated with the musculature, is more marked in inspiration than expiration, and is found most commonly in the lower parts of the back but can be heard over the entire chest. In order to eliminate this, the musculature must be shunted out, which is done during examination of the back by having the subject bend forward in the sitting posture and fixing the auscultated side during ascultation of the shoulder girdle. Pressure with the shell of the phonendoscope also tends to exclude the muscular part of the respiratory sounds. The origin of the harsh muscular respiration is attributed to a number of factors: changes in the tonus of the muscle, shivering during examination, etc. If one is accustomed to eliminate the harsh muscular breathing, it will be found that pulmonary harsh breathing occurs rarely.—*Über das rauhe Atmen (Unreines und muskelrauhes Atmen), A. Winkler, Ztschr. f. Tuberk., July, 1923, xxxviii, 329.*—(*H. J. C.*)

Transmission of Pulmonary Sounds to Healthy Side.—Laennec recognized the essential facts of transmission, accounting for tracheal râles, bronchial râles, metallic tinkle, etc., and he cited facts which showed the possibility of transmission from a distance of these sounds. Trousseau recognized the transmission of bronchial whisper to the opposite side, accompanied by almost normal breath-sounds. The largest number of careful observations, however, have been made by Rilliet and Barthez, and Barthez and Sanné in children. They state that the stethoscopic sounds may produce the illusion of a double lesion. They express the opinion that in the chests of children, the small dimensions of the thoracic cage and thinness of its walls are a factor in this ease of transmission. They distinguish transmitted from autochthonous sounds by the gradual diminution from a fixed point of maximum intensity; usually, also, it is the whispered voice or souffle that is particularly transmitted, dulness and râles being absent. They admit, however, that râles too may be propagated, and Faisans mentions the transmission of sibilant râles. In children enlarged bronchial lymph nodes cause much confusion by giving rise to signs simulating cavities, in various parts of the lungs. Both physicians and surgeons soon noted that auscultation was not sufficient to indicate the exact site of a purulent pleurisy, as exploratory puncture often showed the pus at another level from the signs. It was, however, the widespread employment of artificial pneumothorax that brought into focus the problem of the transmission of sounds. In such cases râles were often encountered which had no counterpart in percussion change or in the radiogram and which were of a similar rhythm and timbre to those on the diseased side. Piéry in 1910 called attention to the frequent misinterpretation of such signs. He noted that direct auscultation in these cases revealed a progressive diminution in resonance of the râles from a given point on the diseased

side. Saugman, Von Muralt and Carpi also published articles along a similar line. Von Muralt called attention to the "symmetrical propagation" of such râles. Dumarest and Murard have referred to the difficulties in diagnosis due to transmitted sounds and to their especial frequency in the interscapular and subscapular regions. Laennec early noted the transmission to a considerable distance of cavernous râles and of certain signs of consolidation. *Methods of recognition of transmitted sounds: 1: Auscultation:* Synchronism and symmetry of adventitious sounds constitute a strong presumption that they are transmitted. Exact similarity is not, however, necessary, for sometimes the râles become finer and drier on the opposite side. It is always the dominating type of râles, the most resonant, that are transmitted, the others being lost in transit. Transmission occurs much oftener in a horizontal than in a vertical plane. The proximity of the vertebral column greatly favors transmission. Certainly transmitted sounds occur much more commonly in the paravertebral region. The propagation of râles from an underlying lesion to the surface does not always follow a direct line. Another diagnostic sign of transmitted râles is their simultaneous disappearance on both sides; this occurs especially in pneumothorax cases. Continuity of transmitted sounds from a given point is not invariable, and Turban has noted silent zones between two similar foci of râles. *2: Palpation:* Fremitus elicited by the hand is regarded by Laennec as a good means of localization. The exaggeration of vocal vibrations corresponds only to a real focus, not to transmitted sounds. *3: Percussion:* This is an important control, as with transmitted sounds there is no dulness unless due to other causes, such as sclerosis or pachypleuritis. *4: Radioscopy and radiography:* These are supplementary and valuable means of determining the presence or absence of underlying lesions. *5: Pneumothorax:* It sometimes happens that following the induction of pneumothorax râles may disappear from the diseased side and appear at a corresponding point on the other. This is believed to be due to the interposition of a cushion of gas between the lung and adjacent chest wall, while continuity remains with the tissues on the opposite side. Again, râles on the sound side are often due to a pushing over of the compressed and diseased lung against the mediastinum, which yields toward the sound side. The resultant sounds, dry or moist râles, tubular or amphoric breathing, are generally most audible near the vertebral column and the hilum. In such cases the diseased lung, although displaced, will usually be found imperfectly collapsed. The media of transmission of sounds may be lymph nodes (Rilliet and Barthez), hepatized zones, cavities and branches of the bronchial tree, and what the authors believe most common, osseous structures, especially the vertebral column.—*La transmission des bruits pulmonaires du côte malade au côte sain, J. Génévrier and R. Daval-Arnould, Rev. d. l. Tuberc., December, 1923, iv, 583.*—(*A. P.*)

Grocco-Rauchfuss Triangle in Diagnosis of Pleurisy.—In exudative pleuritis with copious exudation the Grocco-Rauchfuss triangle is a constant sign. The determination of the triangle does not with certainty indicate the presence of a large exudate, since it can exist when there is present an extensively thickened pleura and when aspiration and roentgenological examination reveal only a slight amount of fluid. In 3 cases of seropneumothorax there was a well-defined paravertebral triangle on the healthy side. Of 17 cases of croupous pneumonia of the lower lobe there was a paravertebral dull triangle on the healthy side in sixteen. The triangle was best defined at the height of hepatization; with lysis it rapidly disappeared although dulness over the diseased lobe persisted for some time. In pneumonia the triangle is less sharply defined and smaller than in exudative pleuritis.—*Über die differentialdiagnostische Bedeutung des "Grocco-Rauchfusschen Dreieckes," A. Lekndorff, Centralbl. f. Inn. Med., April, 1923, xliv, 273.*—(*H. J. C.*)

D'Espine's Sign.—The clinical value of D'Espine's sign is established. It is essential that the observer has had the necessary experience, so as to know the normal range of the bronchophony, namely, in children up to 7 years to the seventh cervical, in the eighth year to the first thoracic, up to the twelfth year to the second thoracic, and up to the fifteenth year to the third thoracic vertebra. To comprehend the tracheal concomitant sound to which is attributed great value, the maplewood stethoscope of Klare has proved serviceable. A critical examination of about 400 roentgen plates revealed a general agreement between D'Espine's sign and the roentgenological findings; in children over 10 years of age there was agreement in 95.3 per cent and in children under 10 years of age 99 per cent It is believed that the whispered voice phenomenon can, under certain circumstances, make unnecessary the roentgen plate. For determining the rare white pulmonary infiltration without hilum involvement, which

frequently does not give physical findings, the determination of the sedimentation rate of the Erythrocytes proved serviceable.—*D'Espinesches Zeichen und Röentgenogramm. Vergleichende Untersuchungen, K. Klare and E. Dehoff, Ztschr. f. Tuberk., July, 1923, xxxviii, 260.*—(*H. J. C.*)

D'Espine's Sign.—D'Espine in his original paper described the sign as a whispering echo following the spoken voice on auscultation over the upper dorsal vertebrae. In a later paper he emphasized the value of the whispered voice saying, "The sign is most obvious if one makes the child speak or count in a low whisper. The voice is then accompanied by an added whispering sound." Fishberg's definition is now generally accepted, the transmission of the tracheal timbre below normal limits when the whispered voice is auscultated. Transmission of the tracheal timbre is indicative of abnormality only when it occurs below the seventh cervical vertebra in young children; below the second dorsal vertebra in children of 12 to 15. These differences are due to the descent of the bifurcation of the trachea with increasing age. It must be emphasized that the mere transmission of vocal resonance does not constitute D'Epine's sign. It is the transmission of the characteristic tracheal timbre which counts. There is a disparity of opinion concerning the value of the sign. Barlow and Thompson attribute an entirely new significance to it. They have found in over 1000 cases that the sign is found in practically every case of early pulmonary tuberculosis, and that it is practically never found unless there are present both constitutional symptoms and X-ray evidence of present or very recent active tuberculosis at the hilum. In other words, D'Espine's sign is an evidence, not of greatly enlarged tracheobronchial nodes, but of hilum tuberculosis. A careful study of properly taken stereoroentgenograms, they assert, shows that D'Espine's sign is present only when there is a mediastinal or central pneumothorax which is, at least in part, at a level which brings it in contact with the trachea. Conversely, a mediastinal or central pneumothorax of any appreciable size usually gives a clear-cut D'Espine's sign. At the State Sanatorium, Meriden, Conn., Carroll and Gibson studied the occurrence of this sign in 411 children, patients in the Sanatorium between 1920 and 1923. They concluded that, since D'Espine's sign occurred in only about 50 out of 411 children, it does not occur indiscriminately. In cases in which X-ray evidence was available 81.6 per cent showed undoubted evidence of hilum disease when judged by a rigid standard. It would, therefore, seem that D'Espine's sign should not be disregarded in the diagnosis of hilum tuberculosis. It should at least be considered as one of the evidences. When the sign is combined with X-ray evidence of abnormality at the hilum, with a positive skin tuberculin test, with manifestations of toxemia not otherwise explained, and when there either is or is not a definite history of exposure to human or bovine tuberculosis—for it must be remembered that "absence of proof of exposure is not proof of absence of exposure"—the diagnosis of hilum tuberculosis is established even in the absence of all other physical signs of abnormality in the lungs. When D'Espine's sign occurs it should be regarded as an urgent indication for the need of other and less easily applied methods of investigation.—*D'Espine's Sign in Hilum Tuberculosis, W. E. Carroll and C. B. Gibson, Boston M. & S. J., February 28, 1924, cxc, 359.*—(*B. T. McM.*)

Anisocoria and Pulmonary Tuberculosis.—The occurrence of dilated pupil on the same side as the lesion in pulmonary tuberculosis has long been mentioned. In 17 per cent of the cases under study the pupil in the same side as the tuberculosis was wide; in no case was there a widening of the pupil on the side opposite to the lesion in unilateral pulmonary disease. Widening of the pupil is not found specifically in lung tuberculosis for it is present in gastric ulcer, pericarditis and probably in cholecystitis, cholelithiasis and splenic tumor. A spontaneous anisocoria does not speak against bilateral pulmonary disease. The spontaneous dilated pupil responds more slowly to atropin than the normal pupil; in the normal pupil dilatation after atropin is at its height in a short time and within five days of the instillation the pupil has returned to normal; the spontaneosly dilated pupil, on the other hand, may still show an atropin effect nine days after introduction of the drug. This sign cannot be used as a differential diagnosis sign between active and latent tuberculosis because it may be present in either active or latent cases. This anisocoria has been thought to be produced by an irritation of the sympathetic system on the side of disease but no anatomic or other evidence is forthcoming to substantiate such a position. Loew's reaction, however, was found to be positive in 6 cases of unilateral pulmonary tuberculosis without anisocoria. This test consists in the instillation of adrenalin (1 to 1000) into each eye. On the diseased side there is dilatation of short duration. It indicates a sympathetic hypersensitiveness. The occurrence of anisocoria in pul-

monary tuberculosis is very interesting from an academic point of view but cannot be regarded as having very great clinical significance.—*Ueber die provozierte Anisokorie bei der Lungentuberkulose, L. Alterthum, Deutsche med. Wchnschr., February 29, 1924, 1, 275.*—(*H. S. W.*)

Inequality of Pupils and Pulmonary Tuberculosis.—The article goes at considerable length into the history of this sign and the question of its value in pulmonary tuberculosis, much having been claimed for it by some clinicians. The author studied 61 cases of unilateral tuberculosis, 21 cases of bilateral tuberculosis and 11 apparently normal cases. Of this number 26 showed inequality of pupils by ordinary examination; mydriasis produced with *euphthalmin* added 45.42 per cent, making a total of 71.42 per cent of cases showing anisocoria. But these results are not to be given undue weight, since the method of producing mydriasis has many faults, and slight errors of technique may cause erroneous results. Of points brought out in the study, and worth citing are the following: An apical lesion is not indispensable for the production of anisocoria; it was found with basal lesions when the apices were normal. Apical lesions do not always cause pupillary inequality. Even with unquestionable anisocoria the underlying cause is not necessarily tuberculosis. Any affection of the lung parenchyma may produce it; bronchiectasis, lung tumor, hydatid cyst of the lung and gangrene of the left base were all observed to occasion it. Finally, the author maintains that pupillary inequalities, excluding those due to corneal lesions or those intrinsic in the eye, may be divided into two groups: the slight and temporary which must be carefully sought out and in which the reflex is intact, and the gross inequalities, marked, permanent, and accompanied often by changes of reflex. Tuberculosis may cause the first like any other pulmonary affection, but it is rarely the cause of the second. Here syphilis plays the preponderant rôle.—*Inégalité pupillaire et tuberculose pulmonaire (inégalité pupillaire spontanée et provoquée), W. Jullien, Ann. d. Méd., February, 1924, xv, 149.*—(*W. B. S.*)

Oculocardiac Reflex in Pulmonary Tuberculosis.—This reflex was first described by Dagnini in 1908 and Ashner in 1909, and many references have since been made to it. The reflex arc comprises the afferent branch of the trigeminal nerve, the organic centres of the vagus or the dorsolumbar sympathetic, and finally the cardiac fibres of the vagus or of the dorsolumbar sympathetic. Thus the reflex may give rise either to cardiac slowing or acceleration, according to which group is dominant. In normal subjects the vagus predominates and causes a slowing of the pulse of 4 to 10 beats. If the parasympathetic vagus centres are particularly sensitive or their antagonists inactive, there may occur a slowing of 12 to 20 beats (exaggerated reflex) or in very marked cases cardiac pauses may be provoked. An insufficient vagus tonus or exaggerated sympathetic stimulation will cause acceleration (inverted reflex). This is encountered in one-fifth of normal subjects. If some portion of the reflex arc is interfered with by any lesion the reflex will be entirely abolished. Opinions differ as to the clinical significance of alteration in this reflex, but it is undoubtedly related to conditions in the autonomic system in some way. A considerable number of observations have been made of it in disorders of the nervous and cardiovascular systems, but few in respiratory affections, especially pulmonary tuberculosis. Pleural effusions have been found to cause an exaggeration during their presence, and asthma a very marked exaggeration, indicative of a vagotonia. Purely cardiac affections appear not to alter the reflex. Toxi-infectious conditions in general, however, abolish it, as does uremia; it reappears after defervescence or detoxication. Scarlatina, according to some, exaggerates it. Secard has noted its frequent abolition in epidemic encephalitis. Endocrine disturbances cause marked alterations, the reflex being exaggerated in hypothyroid states and chromaffine insufficiency, and status thymolymphaticus. It is most often exaggerated in Graves disease, although it may be abolished or inverted. The observations made on tuberculous patients were carried out as follows: The reflex was elicited by digital compression at some time between the digestion periods with the subject reclining. Compression of the eyes was not begun until the pulse registered the same for two successive quarter-minutes. Five seconds was allowed for transmission of the reflex, and the pulsations then counted for a period of fifteen seconds, repeated if the result was uncertain. Of 45 cases, studied, the *R. O. C.* was as follow: Normal in 10, or 22.2 per cent; exaggerated (slowing of 12 to 20 beats) in 11, or 24.2 per cent; strongly exaggerated in 11, or 24.2 per cent; abolished in 10, or 22.2 per cent; inverted in 3, or 6.66 per cent. It is, therefore, concluded that the most commonly met with dystonia in tuberculous patients is hyperparasympatheticotonia (vagotonia) and that hyponeurotonia is of the same frequency as normal tonus, while hypersympatheticotonia

is rare. A correlation of the results with the clinical condition of these patients discloses that the patients with normal reflex, with two exceptions, had very little evidence of toxemia. Those with exaggerated reflex, on the other hand, were generally suffering from well marked toxemia, often associated with rapidly evolving ulcero-caseous lesions. There were also included one case with hypo-ovarian disturbance, one with an Addisonian syndrome with melanoderma, and one with artificial pneumothorax. Tracheobronochial adenopathy may have played a part in some by causing pressure on the vagus. The group with abolished reflex were patients with very advanced disease, more or less cachetic and enfeebled, and in them it is only an indication of terminal anergy.—*Le réflex oculo-cardiaque, chez les tuberculeux pulmonaires, M. Perrin and R. Yovanovitch, Rev. d. l. Tuberc., 1924, v, 46.*—(*A. P.*)

Diagnosis of Tuberculosis.—The importance of the early diagnosis of tuberculosis cannot be too strongly emphasized. Such early diagnosis is not always easy. The fact that a patient has a definite disease other than tuberculosis does not exclude the latter. On making the diagnosis the physician should tell the patient the exact condition in order to elicit his coöperation. The principal means used in making a diagnosis are study of the symptoms, history, physical signs, X-ray, and laboratory findings. These are placed in the order of their relative value. Too much dependence must not be put on the laboratory report. The finding of bacilli is proof of the presence of the disease; if they are not found, it is not proof in itself that the disease is absent. Physical signs and X-ray findings are of little value when taken alone and must be corroborated. The taking of a careful history and an intensive study of the symptoms are of the greatest importance in making a diagnosis of tuberculosis. Besides the family history the habits and occupation of the patient should be carefully gone into. It is best to find out how long it has been since he felt perfectly well rather than how long he has been sick. The symptoms should be studied systematically and for this purpose the grouping according to Po ttenger is favored. The importance of digestive disturbances, loss of weight, rapid pulse, night sweats, and fever is stressed. Temperature, properly taken, is of the greatest value in determining active disease. Hoarseness, cough, pains in the chest and shoulders are given careful consideration. If any symptom is pathognomonic of tuberculosis it is the spitting of blood. Pleurisy also holds an important place in the diagnosis, for the majority of pleurisies are tuberculous and should be treated as such. History of having had pleurisy is second only to that of blood spitting and when either of them has been present in the past, the physician need not have any great fear of making a diagnosis of tuberculosis. If every physician would place more importance on the history and symptoms and use them more extensively, more cases of early tuberculosis would be discovered.—*A Study of History and Symptoms as a Practise Aid in the Diagnosis of Pulmonary Tuberculosis, S. A. Slater, Med. Jour. and Rec., July 2, 1924, cxx, 1.*—(*J. S. W.*)

Histopathological Examinations in Diagnosis of Tuberculosis.—Study of guinea-pig infection shows that latency is of frequent occurrence, and that with the same strain of bacillus the types of reaction may vary greatly in different guinea pigs, some reactions being generalized, others localized in various different regions, some rapidly fulminating, others latent. But notwithstanding these gross differences, conclusive evidence of infection may be obtained by histopathological examination of the several tissues made at a suitable time—one month to six weeks after inoculaiion. A uniform technique for handling all inoculated animals has been developed in the Corps Area laboratories: suspected material is injected subcutaneously into the region of the left superficial inguinal lymph nodes; a liberal amount of material (1 to 2 cc.) is inoculated. Six weeks later all animals are autopsied irrespective of gross appearances. Tissue is taken from both lungs, both kidneys, spleen and liver; if any lymph nodes are unusual they are sectioned; other organs showing gross changes are studied. No animal is pronounced nontuberculous on gross findings alone. These studies have led to the conclusion that many guinea pigs ordinarily classed as negative may be shown to be infected, and vice versa. It follows that many chronic conditions really tuberculous have often remained unrecognized because of inadequate examinations. Material for examination, either from inoculated animals or from patients, may be forwarded to the Corps Area laboratories or to the Army Medical Museum. In the preparation of material only the ordinary routine need be employed, but a liberal amount of the suspected material should be sent.—*Tuberculosis in Chronic Inflammation, E. R. Scott and R. W. French, Military Surgeon, January, 1924, liv, 91.*—(*E. C.*)

Diagnostic Criteria of Pulmonary Cavities.—Before the discovery of the

roentgen ray pathologists were well aware of the frequency of cavities not suspected during the life of the patient. The failure to recognize these cavities is due in part to the great depth or to the slight extent of the lesions which gives no physical signs; certain other lesions are masked by inflammatory reactions of the surrounding structures, such as consolidation, effusion or pleural adhesions; also a considerable number of cavities are enclosed in an area of condensation or of infiltration, the signs of which conceal those properly ascribed to excavation. Since the development of radiology, it has been learned that a goodly number of these inaudible lesions are visible on the screen or film and that "silent" cavities are just as frequent as the autopsies of former days showed them to be. Radiology, of course, is not as accurate as autopsy studies, and radioscopy less so than radiography. Nevertheless, it is of great value to take and study X-ray pictures at intervals during the illness of the tuberculous patient, and especially to take a picture a few days before death or after death before the autopsy, finally comparing the findings with those of the postmortem examination. Such studies prove that there are silent cavities which are visible, but one must guard against the abuse of this fact, when such X-ray findings are not controlled by necropsy, for there are false cavity shadows. The method of Sicard and Forestier, of using intratracheal injections of *lipoidol* may throw some light on this question. There are also silent cavities which are visible, and cavities giving rise to sounds but which are invisible because of other densities. Certain invisible cavities may be suspected from the presence of their draining bronchus which sometimes is definitely outlined, leading to the clouded area which masks the image of the cavity. Considering these facts, it would be a mistake to emphasize the failure of auscultation and the superiority of radiology. The incontestability of the existence of silent cavities is proved by references in the literature, some of which are cited, as is a case illustrating this phenomenon. Three necessary conditions for the recognition of silent, though visible, cavities are acuteness of hearing of the examiner, proper technique of exploration, and the demonstration of the cavity at autopsy. Clinicians sometimes report cavities as silent when in reality they give audible signs; this error is made when, as often happens, the cavity is situated at the lateral margin of a lobe close to a fissure. These cavities cast clearly defined radiological shadows but may give no stethoscopic signs if one auscultates only the anterior and posterior regions of the thorax; if, however, the axillary region along the course of the interlobar fissures is auscultated positive signs are almost always heard. This is more true of cavities than of any other pulmonary lesion. Regarding the third condition, no one has the absolute right, even with the aid of the X-ray to affirm the existence, much less the extreme frequency, of silent and visible cavities unless he can prove this extreme frequency at autopsy. One cannot deny that false cavity shadows may be just as frequent, especially if a radioscopic examination is done, since, by changing the position of the subject under the screen the dissociation of the false circle can often be demonstrated. Besides false shadows and truly silent cavities, attention is directed to a group of cavities which are not silent but which give signs that cannot be called classical. In suggesting a revision of our conceptions regarding signs of cavity, two ideas are stressed: *1:* Cavity signs may exist where there is no cavity; this is noted in large pleural effusions when signs arise from the large bronchi remaining patent in the collapsed lung, as well as in cases with deviation of the trachea, in certain forms of pleurocortical tuberculosis, and in certain cases of encysted pneumothorax. *2:* A cavity may reveal itself by signs other than those called "cavitary." The ideas of Burnand and Carrard, of Ameuille and Gally, and of Dumarest, bearing on this point are reviewed. A case record is included showing a cavity which cast an X-ray shadow and gave rise to intermittent symptoms, but the only stethoscopic sign of which was a localized area of distant bubbling râles heard in the upper axilla only when the roentgenograph showed a small collection of fluid in the cavity. From this observation it is deduced that "silence" of a cavity may not be constant, but intermittent; furthermore, the "stagnant" condition of a lesion which is nonprogressive, and about which the parenchyma is not diseased, may explain the habitual absence of classical cavity signs, especially if the drainage of the cavity is poor, as evidenced by variations in the bacilliferous expectoration. This poor drainage of a fistulous, narrow, perhaps tortuous tract, in addition to other anatomic factors, such as the absence of consolidation about the cavity, and the rigidity of the cavity wall which does not yield during coughing, produces peculiar physical conditions which explain the "silence," or at least the absence of classical cavity signs. The circulation of air is retarded and reduced to a minimum by the narrowing and the sinuosity of the draining bronchus; and the transmission of the cavity murmur, already feeble on this account, is further reduced

because of the absence of consolidation about the cavity. It is important, therefore, always to correlate the findings from different methods of examination. General symptoms and the character of the expectoration may indicate the presence of a cavity; it is the function of the stethoscopic and radiological examination to localize this cavity. The cavity may actually be silent, but to determine this point one must make sure that there exists no adventitious sign, not even a consonating râle, and that the "silence" is constant and not merely intermittent. If the "silence" is absolute and constant and the cavity is revealed only by the radiological image, one must prove that it is not a question of a false cavitary image or a small partial pneumothorax. *Conclusions:* 1. The stethoscopic finding of classical signs of cavity does not necessarily imply the existence of a cavity; these signs may exist without cavity (pseudocavity stethoscopic signs). 2. The radiological findings of a cavity shadow does not necessarily imply the existence of a cavity; there are false cavity shadows. 3. There are true cavities which are silent and visible; but these really silent cavities are, perhaps, less frequent than is actually believed. The truth must be sought in the revision of signs designated "cavitary;" this revision will probably show that stethoscopic and radiological examinations, far from being in frequent disagreement and conflict, will contribute, if correlated methodically, to define accurately the complete semeiology of cavitary signs.—*Notions nouvelles apportées par l'exploration radiologique au diagnostic des cavernes pulmonaires, Étude critique des cavernes muettes, E. Sergent, Presse Méd., June 11, 1924, no. 47, p. 509.—(J. B. A.)*

Demonstration of Cavity by Transparietal Injection of Lipiodol.—In a case, in which clinical signs were absent and radioscopy was indecisive, Ameuille was able to demonstrate the existence of cavity. The opaque liquid sank to the bottom and a cough sent it toward the hilum, and later out through the trachea, after having previously overflowed into the bronchi of the better side.—*Mise en évidence d' une caverne par l'injection transparietale de lipiodol, Méchanism de l'infection et de la défense du poumon sain, Ameuille, Meeting, Soc, Méd. d. Hôp. d. Paris, May 30, 1924. reported in Presse Méd., June 4, 1924, no, 45, p. 493.—(A. P.)*

Differential Diagnosis of Miliary Tuberculosis and Secondary Lung Tumors.—Pulmonary metastases of malignant tumors may bear a close resemblance to miliary tuberculosis in the roentgen picture. Four cases are described (2 of which were previously reported by other authors) in which this was the case, including a chorioepithelioma and a melanosarcoma in the author's experience, and one case each of Boeck's disease and gastric carcinoma in the experience of others. In the case of the chorioepithelioma the round nodules seen in the X-ray plate were slightly larger than miliary tubercles, increased in number from above down and decreased from the hilum out. This arrangement and appearance were sufficient to exclude tuberculosis in the diagnosis. The differences from miliary tuberculosis in the case of the melanosarcoma were similar.—*Zur Differentialdiagnose miliarer Lungenprozesse und sekundärer Lungentumoren, J. Blum, München. med. Wchnschr., April 25, 1924, lxxi, 545.—(E. R. L.)*

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